VOLUME 38 NUMBER 1, PART I JULY, 1954

# AMERICAN JOURNAL
## OF
# OPHTHALMOLOGY

THIRD SERIES FOUNDED BY EDWARD JACKSON

CONTENTS


Endocrine exophthalmos ..... *Frederick C. Cordes* 1

A new trephining operation .... *Frederick H. Verhoeff and Paul A. Chandler* 21

Argyll Robertson pupil ..... *Howard A. Naquin* 23

Unilateral Argyll Robertson pupil ..... *Julia T. Apter* 34

Bilateral homonymous hemianopsia ..... *Frederic M. Reese* 44

Diagnosis in exotropia ..... *Martin J. Urist* 58

Postnatal cataracts in prematures ..... *Loren P. Guy* 65

Investigation of eye in monochromatic light ... *M. Hultin and T. Kornerup* 68

Loss of the lower fornix ..... *Alston Callahan* 75

Retrobulbar injection of Xylocaine® ..... *Dan A. Russell, Jr., and Jack S. Guyton* 78

Trypsin in ophthalmology ..... *Joseph M. Hopen* 84

Clinical pathologic conference ..... *Parker Heath* 88

Magnetic foreign body ..... *Gregory Flynn and Morgan Raiford* 90

Orbital retractor ..... *Robert L. Alexander and Peter H. Ballen* 91

Orbital implant ..... *H. Arruga* 93

Office treatment tray ..... *Ralph J. Carbo, Jr.,* 95


DEPARTMENTS


Society Proceedings 97 Correspondence ... 111 Abstracts ..... 114

Editorial ..... 109 Book Reviews ..... 111 News Items ..... 127


For complete table of contents see advertising page xxiii

Publication office: 450 Ahnaip St., Menasha, Wisconsin

*Copyright, 1954, Ophthalmic Publishing Company, 664 North Michigan Avenue, Chicago 11, Illinois*

***Subscription price in United States twelve dollars yearly. In Canada and foreign countries fourteen dollars.*** Published monthly by the Ophthalmic Publishing Company. Subscription and Advertising Office: 664 North Michigan Avenue, Chicago 11, Illinois. Entered as second class matter at the post office at Menasha, Wisconsin. Printed in U.S.A.

# *Complete* ARTIFICIAL EYE *Service*

Experience gained through over 100 years of artificial eyemaking enables us to produce and fit the very finest in artificial eyes. Glass and plastic eyes made to order or available from stock. Eyes fitted to all types of motility implants.

**fitting of artificial eyes through the mail**

- Orders filled the same day received
- Eyes sent on memorandum
- Liberal assortment of either glass or plastic
- Samples accurately matched
- Superior quality eyes—Finest workmanship

*Serving the Profession Since 1851*

## *Mager and Gougelman, inc.*

30 NORTH MICHIGAN AVENUE • CHICAGO 2, ILLINOIS

DETROIT • CLEVELAND • KANSAS CITY • MINNEAPOLIS • NEW ORLEANS • ST. LOUIS
NEW YORK • BOSTON • BUFFALO • PHILADELPHIA • PITTSBURGH • WASHINGTON

Gonioscopic lens over cornea may be used to distinguish between wide- and narrow-angle glaucoma.

· *Potent aid in wide-angle glaucoma*

**FLOROPRYL®**

(ISOFLUROPHATE, MERCK)

FLOROPRYL is particularly useful when other miotics fail. One instillation of FLOROPRYL per day is frequently adequate for controlling intra-ocular tension with virtual freedom from systemic effects.

**SUPPLIED:** As a 0.1 per cent solution in peanut oil, 5-cc. vials.

**BAUSCH & LOMB**

# Professional Ortho-Rater

**. . . A NEW PRECISION TESTING INSTRUMENT FOR PROFESSIONAL MEASUREMENT OF VISUAL SKILLS**

Visual skills of more than 12,000,000 workers have been tested through the Bausch & Lomb Occupational Vision Service. Of these, about 42%—more than 5,000,000—have been found to be in need of professional eye care.

Now the same tests of visual performance can be made in your office. Use the Bausch & Lomb Professional Ortho-Rater for pre-refractive and post-refractive evaluation of the twelve visual skills. And for referred Occupational Vision patients it provides objective evidence of improvement resulting from professional care.

BAUSCH & LOMB

SINCE B&L 1853

# Cortef*
# for inflammation, neomycin for infection:

## 1. Neo-Cortef* *ointment (topical)*

*Each gram contains:*

Hydrocortisone acetate ..... 10 mg.
(1%) or 25 mg. (2.5%)
Neomycin sulfate ..... 5 mg.**
Methylparaben ..... 0.2 mg.
Butyl-p-hydroxybenzoate ..... 1.8 mg.

*Supplied:*
5 Gm. and 20 Gm. tubes in plastic cases.

## 2. Neo-Cortef* *ophthalmic ointment*

*Each gram contains:*

Hydrocortisone acetate .. 15 mg. (1.5%)
Neomycin sulfate ..... 5 mg.

*Supplied:* 1 drachm applicator tubes

## 3. Neo-Cortef* *ophthalmic drops*

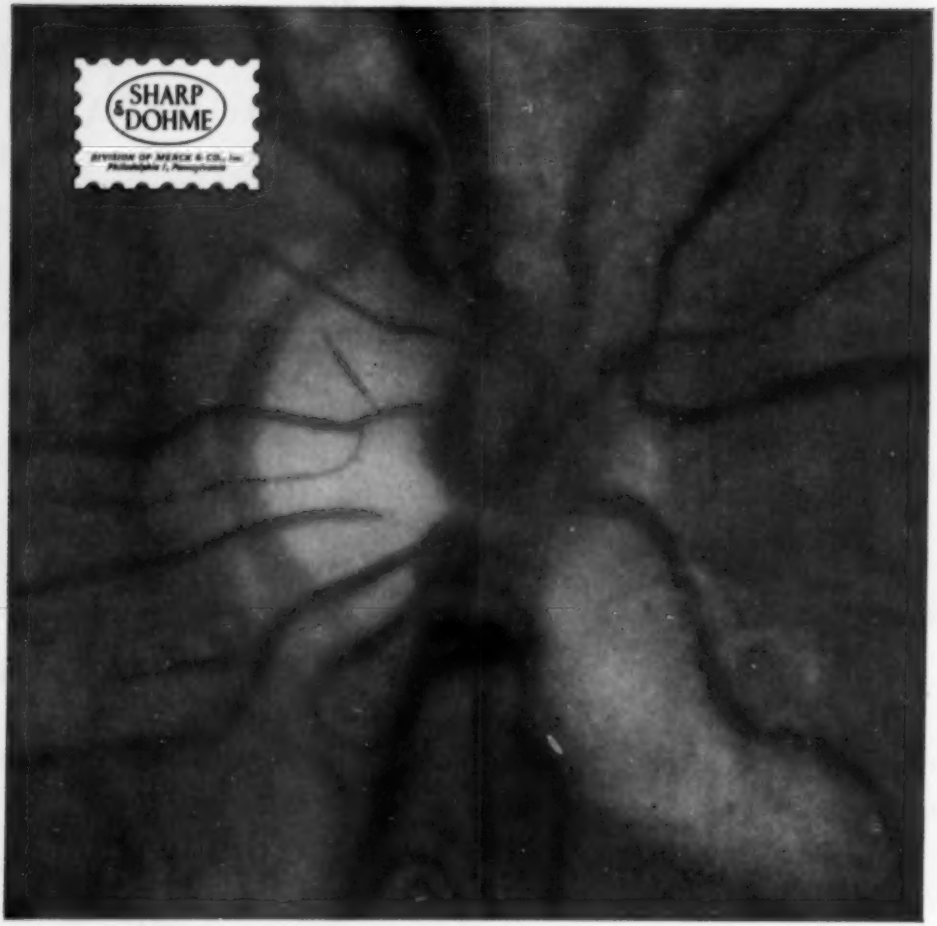
*Each cc. contains:*

Hydrocortisone acetate .. 15 mg. (1.5%)
Neomycin sulfate ..... 5 mg.**

*Supplied:* 5 cc. dropper bottles

*TRADEMARK
**EQUIVALENT TO 3.5 MG. NEOMYCIN BASE

THE UPJOHN COMPANY, KALAMAZOO, MICHIGAN




**Consider its advantages in posterior segment disease**

*HydroCortone*®

(HYDROCORTISONE, MERCK)

**CLINICAL HIGHLIGHTS:** Hydrocortisone is a principal adrenocortical hormone and the steroid with the most potent anti-inflammatory action. In smaller doses, HYDROCORTONE produces the same therapeutic effect as cortisone. Systemic therapy with HYDROCORTONE Tablets usually permits improved management of posterior segment eye disease and of anterior segment disease unresponsive to topical therapy.

Nonspecific choroiditis, uveitis, iridocyclitis, chorioretinitis, retinitis centralis, sympathetic ophthalmia, ophthalmic herpes zoster (not herpes simplex) and optic neuritis have all responded favorably to systemic steroid therapy.

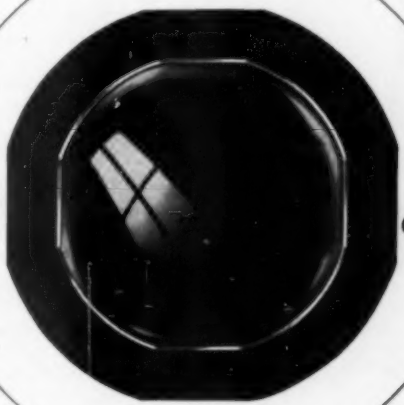
**SUPPLIED:** ORAL—HYDROCORTONE Tablets: 20-mg., bottles of 25 tablets; 10-mg., bottles of 50 and 100 tablets; 5-mg., bottles of 50 tablets.

**All HYDROCORTONE Tablets are oval-shaped and carry this trade-mark:**

FOR FAITHFUL INTERPRETATION OF YOUR *Rx*

You can depend on Shuron Widesite Corrected Curve Lenses for faithful interpretation of your prescription . . . because they are always and only First Quality. And your patients see as nature intended them to see . . . with marginal correction providing clear, undistorted vision from edge to edge.

WIDESITE



CORRECTED CURVE LENSES

Surfaces are polished to mirror finish . . . free from aberration. No seeds, bubbles, striae or other internal flaws. Correctly centered . . . precision curves . . . exact powers . . . accurately controlled thickness. Shuron's Widesite Lens program coincides with the optical industry's objective of eliminating all but corrected curve lenses.

Shuron Optical Co., Inc.
Geneva, New York

7754-2

Specify SHURON for First Quality

# NOW

*by the Makers of America's finest Professional Instruments*

## The MOTORIZED CHAIR

**NEW PRODUCT.** Now! Greater ease and comfort for both doctor and patient with AO's new motorized chair. Here, after years of careful development by our design engineers, is a motorized chair backed by a name you know and trust. A mere touch of your toe brings the chair exactly into position.

**NOT AN ADAPTATION.** The new motorized base unit is an original design created and developed by American Optical. The design is unique and desirable in that the powerful and quiet motor and pump assembly is completely enclosed within the chair's streamlined base housing. There are no clumsy projections at the base of the chair.

**OTHER FEATURES.** The chair's base ring can be interlocked with that of the AO instrument stand, achieving greater neatness and rigidity. Head rest, arm rests, back and seat, adjust to any desirable position. The chair rotates, regardless of the position of the seating unit, and locks anywhere. You'll find the controlling pedals all conveniently placed. The air foam rubber seat and the back rest are upholstered in genuine leather. Choice of three popular standard colors—the new Jade Green, Ivory Tan, or Grained Mahogany.

**ASK TO SEE IT.** You owe it to yourself to witness the outstanding performance features of AO's newest product.

American Optical

INSTRUMENT DIVISION

BUFFALO 15, NEW YORK

NOW!

relief

even for the refractory patient...

in nasal or ophthalmic allergies

# CORTICLORON

(CORTISONE plus CHLOR-*TRIMETON*®)

**STERILE SUSPENSION**

15 *cc. STERILE* DROPPER BOTTLE

anti-inflammatory · anti-allergic · decongestant

safe for the hypertensive patient

CORTICLORON® Suspension (brand of cortisone acetate and chlorprophenpyridamine maleate).
®T.M.

Schering

CORTICLORON

nasal or ophthalmic

**RAPID ACTION** **RAPID ACTION**

**RAPID RECOVERY** **RAPID RECOVERY**

**A TIME SAVER IN CYCLOPLEGIA AND MYDRIASIS**

A new, virtually non-irritating cycloplegic-mydriatic compound with marked advantages in rapidity of action (30-60 minutes), depth of depression of accommodation, and promptness of recovery ... 12-24 hours *without* a miotic ...

Now ... Council Accepted

# CYCLOGYL®

Hydrochloride

brand of CYCLOPENTOLATE hydrochloride

Effective in cases of darkly pigmented eyes normally refractory to cycloplegics,[1] CYCLOGYL Hydrochloride does not appreciably affect intra-ocular pressure, and may be used with safety in all age groups. It "may successfully replace, or will certainly be a useful adjunct, to the use of homatropine and even atropine for routine office refraction."[2]

*Write for literature*

**CYCLOGYL** (Hydrochloride) Available in 0.5% and 1.0% Solution, in 15 cc. Bottles; 1.0% Solution, in 2 cc. Bottles

*Schieffelin & Co.*, pharmaceutical and research laboratories since 1794

18 Cooper Square, New York 3, N. Y.

1. Gettes, B. C., and Leopold, I. H.: A. M. A. Arch. of Ophth. 49:24 (Jan.) 1953.
2. Stolzar, I. H.: Am. J. Ophth. 36:110 (Jan.) 1953.

*New Design . . . and Performance*

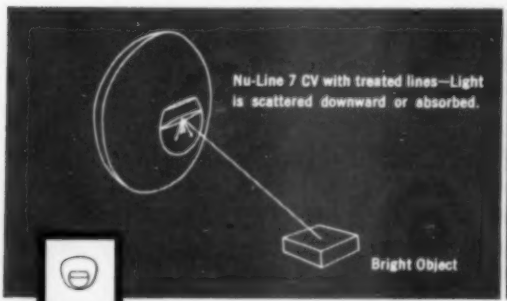
**nu-line® 7 CV**
TRADE MARK
*patent pending*

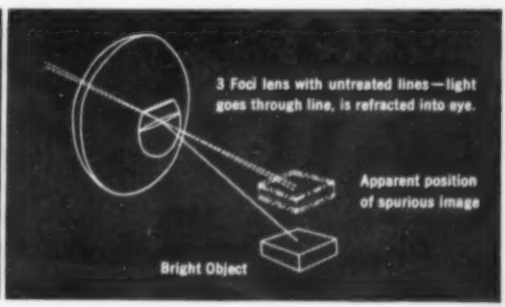
The NU-LINE 7 CV is designed to outperform *any* multifocal lens of 1.75 D. add or higher.

[1] Incorporates *all* the superior advantages of the Univis CV 3 foci lens, plus:
[2] Processed segment lines reduce false images (white or multicolored) over 80% by photo interceptor cell test.
[3] Better cosmetically—processed segment lines blend into facial coloring.
[4] Positive Univis Identification from processed lines.
[5] Popular segment sizes—7 mm intermediate and 23 mm segment width.

***Etiology.*** For the past three years Univis Continuous Vision Lens performance has been guaranteed. Patient satisfaction under that guarantee has exceeded 97%. Striving for perfection, Univis conducted clinical analyses on the less than three percent that were returned. It was clearly established that intruding light images (white or multicolored) were the chief cause of patient complaint. These images were thought by some to be the result of chromatic aberration in the segment glass. This was unfounded because Univis, like other better manufacturers, has been using a color-free segment glass for many years. It was conclusively proven that these annoying images resulted from reflections or refractions by *segment lines.*

***Performance.*** Univis Researchers have answered this problem with an entirely new lens. By actual photo interceptor cell test, the processed segment lines of the NU-LINE 7 CV absorb or diffuse over 80% of light striking the lines. *(See charts below)*



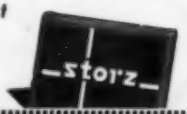


3 Foci lens with untreated lines—light goes through line, is refracted into eye.
Apparent position of spurious image
Bright Object

Literature available on request

The Univis Lens Company / Dayton • New York • Chicago
Los Angeles • San Francisco • Philadelphia

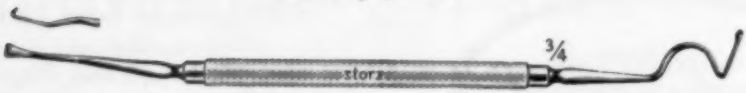
storz Surgical Instruments

*Ready for mailing . . . 1954 Issue of Storz Eye Instrument Catalog showing newest developments.*




## *New!* Iris Retractor and Lens Expressor for Round Pupil Cataract Surgery

A. Benedict Rizzuti, M.D.
Brooklyn, N.Y.




*Described in June issue*

## *New!* BALLEN-ALEXANDER ORBITAL RETRACTOR

*Large and Small*

*Described elsewhere in this issue by:*

ROBERT L. ALEXANDER, M.D.
Sacramento, California

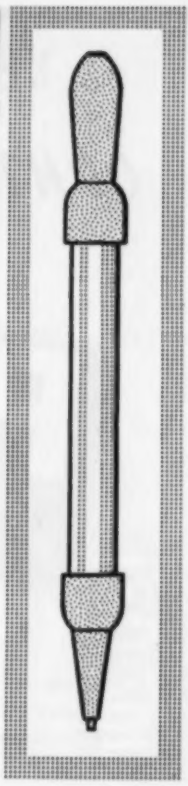
*Storz Instrument Company,* 4570 AUDUBON AVE., *St. Louis 10, Mo.*

# now available!

# OPHTHALMOS

sterile, stable, eye medications in the new

# steridroppa*



Single dose sterile eye solutions
in disposable dropper units, developed by
Robert R. Feinstein and Associates in collaboration with major eye hospitals in the U.S.A.
A major advance in the maintenance of sterility in operating procedure and post operative therapy. A necessity in every eye operating room and wherever eye injuries are treated.
STERIDROPPAS contain all important solutions required for ocular surgery.

* trade mark
pat. pend.

write now for further details to

OPHTHALMOS, INC.
4808 BERGENLINE AVE.
UNION CITY, N. J.

Just pin this to your letterhead and mail
Please Send Details on
Ophthalmos
Steridroppa
Solutions

# A NEW BufOpto STERILE BUFFERED OPHTHALMIC SOLUTION

# PILOCEL 1% & 2%

**EFFECTIVE IN GLAUCOMA**

**PILOCEL 1%** A sterile buffered methylcellulose solution containing Pilocarpine Hydrochloride 1%.

**PILOCEL 2%** A sterile buffered methylcellulose solution containing Pilocarpine Hydrochloride 2%.

**ADVANTAGES:**

Pilocel provides the physician with a sterile, accurately prepared miotic. The methylcellulose base insures more intimate and prolonged contact with the eye. Highly effective . . . well tolerated . . . convenient to use.

**PACKAGE:**

Pilocel solutions are packaged in 10 cc. hermetically sealed aluminum capped amber vials, dropper enclosed.

**WRITE FOR SAMPLES & LITERATURE**

*Pilocel — Brand of Pilocarpine Hydrochloride.

**Other Ophthalmic Solutions**
**Bufopto with Neozin**
**Bufopto with Methulose**
**Bufopto with Sulfacel-15**
**Bufopto with Zinc Sulfate**

AVAILABLE AT ALL PRESCRIPTION PHARMACIES

**PROFESSIONAL PHARMACAL CO.**
INCORPORATED
PHARMACEUTICAL MANUFACTURERS
San Antonio, Texas

*drop for drop...*

*and minim for minim...*

# ISO-SOL®

STERILE OPHTHALMIC SOLUTIONS

*assure the utmost in:*

- CLINICAL EFFECTIVENESS
- PATIENT PROTECTION

*Scientifically prepared,*
ISO-SOL ophthalmic solutions are:

- physiologically active
- exceptionally well tolerated
- buffered for stability
- sterile and bacteriostatic

*and* available in a variety of preparations routinely employed in ophthalmologic practice.

**Specify ISO-SOL— each dropper bottle is sterile, sealed and dated.**

*Now Available...*

**for the pilocarpine-sensitive patient***

**CARBACHOL CHLORIDE, U.S.P. 0.75%**

*in a potent, non-sensitizing, non-irritating miotic in a low surface tension vehicle to assure smooth, consistent and enhanced penetration with a minimum of undesirable side effects. Available in 7.5 cc. sterile, sealed bottles.*

* Morrison, W. H.: Am. J. Ophthalmol. 37: 391, 1954.

Write for a descriptive list of all ISO-SOL products.

MYOTICS
MYDRIATICS
ANTISEPTICS
LUBRICANTS

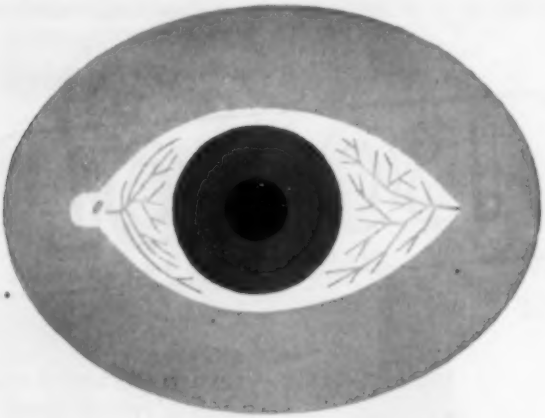
THE ISO-SOL® COMPANY, INC.

130 FLATBUSH AVENUE
BROOKLYN 17, N. Y.

*pioneer specialists in sterile ophthalmic pharmaceuticals*

IN CANADA: RANAR CO., 1245 ST. CATHERINE ST. W., MONTREAL

to
control
ophthalmic
infections...



# Furacin® Ophthalmic Liquid and Ointment

brand of nitrofurazone, Eaton

Furacin combines powerful antibacterial action against a wide range of gram-positive and gram-negative organisms with remarkable lack of irritation. The patient feels no stinging sensation, and regeneration of the highly sensitive corneal epithelium is not retarded. Furacin is effective in the presence of pus and mucus, and remains stable at body temperature.

**INDICATIONS:** [1,2,3,4] Conjunctivitis (especially staphylococcic) • Blepharitis and Abscesses of the Lids • Infected Corneal Ulcers • Keratitis • Dacryocystitis

**FORMULAS AND AVAILABILITY:**

**Furacin Ophthalmic Liquid** contains Furacin 0.02% dissolved in isotonic aqueous solution. ½ fl. oz.

**Furacin Ophthalmic Ointment** contains Furacin 1% in a petrolatum type base. ¼ oz. tube.

**References**

1. Pritikin, R. I.: J. Internat. Coll. Surgeons 17: 234, 1952.
2. _____, and Duchon, M. L.: Mil. Surgeon 109: 706, 1951.
3. _____, _____, and Farmer, H. S.: Mil. Surgeon 108: 309, 1951.
4. Brennan, J. W.: Am. J. Ophth. 35: 1343, 1952.

rapidly
effective
nonirritating

EATON LABORATORIES Inc.
NORWICH, NEW YORK

THE NITROFURANS—A UNIQUE CLASS OF ANTIMICROBIALS
PRODUCTS OF EATON RESEARCH

# DROP-TAINER

ALCON'S NEW PLASTIC DROPPER CONTAINER

**ADVANTAGES**
**OF Alcon DROP-TAINER**
**Ophthalmic Solutions: Buffered — Sterile — Stable; Preserved against contamination; Better "drop" control; Convenient to use; Uniformly the same wherever obtained; Economical — Unbreakable — No loss from spillage; Solutions last up to twice as long.**

***Sterile buffered ophthalmic solutions NOW AVAILABLE in 15cc DROP-TAINER:***

**Isopto® Atropine 1% • Isopto® Homatropine 2% • Isopto® Hematropine 5% • Isopto® Hyoscine HBr. .25% • Isopto® Phenylephrine 2% • Isopto® Phenylephrine 10% • Isopto® Eucatropine 3% • Isopto® Carpine .5% • Isopto® Carpine 1% • Isopto® Carpine 2% • Isopto® Carpine 4% • Isopto® Eserine .25% • Isopto® Eserine .5% • Isopto®-Frin.**

**("Isopto"® designates Alcon's sterile ophthalmic solutions containing Methyl Cellulose.)**

***DROP-TAINER sterile ophthalmic solutions without Methyl Cellulose:***

**DROP-TAINER Butacaine 2% • DROP-TAINER Tetracaine HCl .5% • DROP-TAINER Phenacaine HCl 1% • DROP-TAINER Zincfrin®* • DROP-TAINER Op-thal-zin* • DROP-TAINER Fluorescein Sod. 2% • DROP-TAINER Benzalkonium Chloride 1:5,000.**

***Zincfrin and Op-thal-zin available also in regular 15cc blue dropper bottles. Please specify DROP-TAINER if the new plastic container is preferred. NOTE: DROP-TAINER label printing removable with acetone. ORDER FROM YOUR PHARMACY. Prescription pharmacies have Alcon products or may quickly obtain them from their local wholesale drug supplier anywhere in the U.S.A. Write for descriptive literature and prices of DROP-TAINER sterile ophthalmic solutions.***

**Alcon**
LABORATORIES, INCORPORATED
FT. WORTH, TEXAS

# THE "PANTOSCOPE"
# THE UNIVERSAL OPHTHALMOSCOPE

FEATURES

- Direct or Indirect ophthalmoscopy by polarised, red-free or natural light.
- Intense 12w. light.
- Variable control for projected slit examination of cornea & anterior chamber.
- Wide-angle field with high myopes.
- Lenses plus 30D to minus 50D.
- Illuminated dial with presbyopic viewer.

**MULTI-PURPOSE EQUIPMENT**

**AUTOFLEX POWER SUPPLY:** **The Universal Unit.**

Automatic extension or retraction of 3 & 12v. cords eliminates untidiness. Additional 3, 6 or 12v. output panel for any instrument taking up to 1 amp. Rheostat brightness control.

keeler

**KEELER OPTICAL PRODUCTS, INC.**

617 S. 52nd St., Philadelphia 43, Pa.

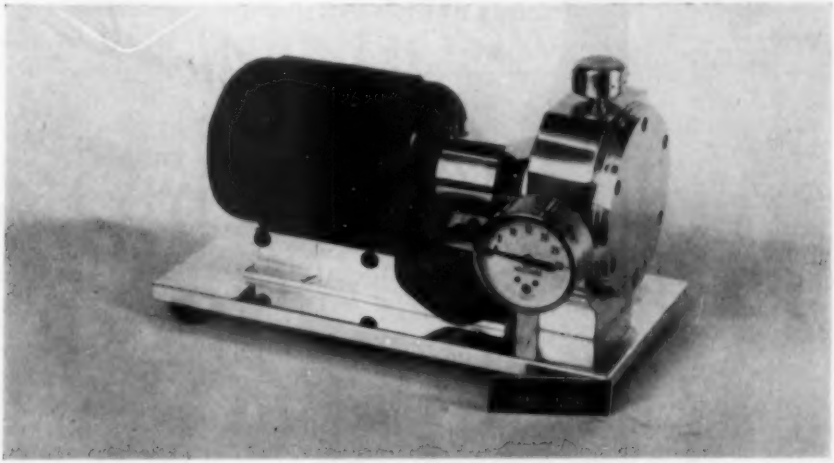
GRANITE 4-5310

keeler

***Please write, phone or call for further details and literature.***

*It is Here:*

# THE BERLINER CATARACT VACUUM PUMP



**This perfectly safe and handy instrument has been designed by Dr. Milton L. Berliner, of New York City on the principle of Dr. Barraquer. It has been received with great enthusiasm by many leading surgeons. The amount of suction is easily adjustable to the desired volume. Each instrument is individually constructed and requires practically no maintenance. The metal parts are either aluminum or stainless steel. The instrument is completely silent and vibrationless. AC or DC may be used. Delivered in sturdy, handsome carrying case.**

*Trade in Allowance for Poser and Universal Lamps*

*The Unique*

## GOLDMANN SLIT LAMP

- **One Arm Control**
- **Hruby Lens for Fundus Examination**
- **Many Other Advantages**

**U.S. Agents Also for:**

**Perimeters, Ophthalmometers and Other Ophthalmological Equipment.**

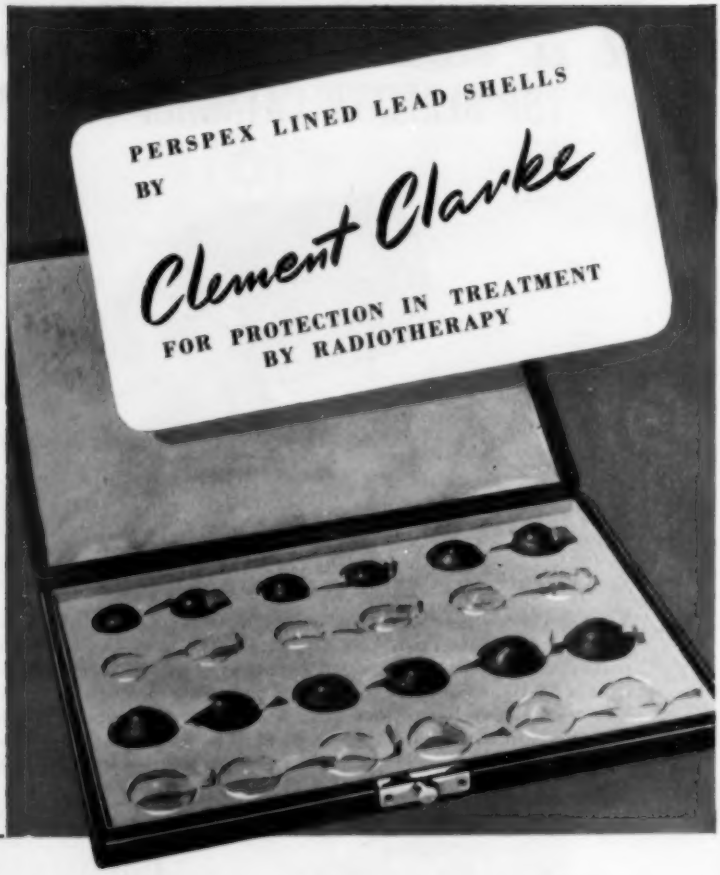
**Can Be Mounted on B & L or AO Stands.**

***Visit our exhibit at the International Meeting, Booths #58, 59, 68 and 69.***

## ALFRED P. POLL

***Ophthalmic Instruments of Top Quality***
**40 West 55th Street, New York 19, N.Y.**

Pat. No. 530108



Perspex lined lead shells in the form of a contact lens afford the eye complete protection when treating parts very close to the eye with X-rays or rays from radio-active substances. By drilling a small hole in the shell it is even possible to carry out localised treatment of a lesion on the sclerotic without exposing the cornea and other parts of the sclerotic to the harmful effects of the radiations. The set of lead shells illustrated comprises six sizes for both right and left eyes. Smaller cases and single shells are also available. *Price, complete in case—$97.30.*

CLEMENT CLARKE

*of England* · 63 WIGMORE STREET, LONDON, W.1

THEODORE HAMBLIN LTD
DISPENSING OPTICIANS
15 WIGMORE STREET
LONDON.W.1.

OPTICIANS BY APPOINTMENT TO H.M. QUEEN ELIZABETH THE QUEEN MOTHER

OPTICIANS BY APPOINTMENT TO THE LATE QUEEN MARY

# Craftsmen-built Ophthalmic Instruments by Hamblins of England

including the World-famous
Lister-Morton Ophthalmoscope
will be shown at the XVII,
INTERNATIONAL CONGRESS OF OPHTHALMOLOGY on September 12th to 17th,
and at the meeting of the
AMERICAN ACADEMY of OPHTHALMOLOGY & OTOLARYNGOLOGY on September 19th to 24th,
(both held at the
WALDORF-ASTORIA, NEW YORK,)
by ANTON HEILMAN of
75, Madison Avenue, New York.

BOOTH NUMBER 107.

# YOU ARE INVITED

## XVII INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

**MONTREAL September 10-11**

**NEW YORK September 13-17**

## CANADIAN OPHTHALMOLOGICAL SOCIETY

**MONTREAL September 9**

## AMERICAN ACADEMY of OPHTHALMOLOGY and OTOLARYNGOLOGY

**NEW YORK September 19-24**

**BERMUDA September 25-30**

# TO ATTEND ALL THREE

Meet distinguished ophthalmologists of the world.
More than one thousand advance registrations.

Write the New York Visitors and Convention Bureau, 500 Park Avenue, New York 22, for reservations at your favorite hotel.

For information: G. Stuart Ramsey, M.D., Physical Sciences Centre, McGill University, Montreal 2, Canada

or

William L. Benedict, M.D., 100 First Avenue Building, Rochester, Minnesota.

# ESTIVIN®

## Eases Ocular Irritation In Hay Fever...

TIVIN is a soothing eye drop which promptly
d effectively relieves ocular and nasal
comfort due to

y fever

ergic conjunctivitis

njunctivitis due to
smoky, dusty, irritating air
presence of foreign bodies
the common cold

TIVIN is an aqueous infusion of "rosa gallica L."
It is decongestive and soothing to
inflamed ocular and nasal membranes.

TIVIN is non-toxic — safe.

sage: One drop of *Estivin* in each eye two (2) or three (3) times daily will alleviate discomfort, and relieve severe itching of the ocular and nasal membranes.

pplied: 0.25 fl. oz. DROPAK*
The DROPAK is a new disposable plastic container which delivers single accurately measured drops of *Estivin*.
**Also supplied:** 0.25 fl. oz. bottle complete with eye dropper

**NEW PORTABLE ONE-DROP "DROPAK"**

ND FOR OFFICE SUPPLY OF *ESTIVIN* IN NEW "DROPAK"

*Schieffelin & Co.*

*trademark

Schieffelin

**Pharmaceutical and research laboratories**
**20 Cooper Square, New York 3, N. Y.**

# a scientific corner

FOR INFORMAL DISCUSSION OF YOUR OPTICAL PROBLEMS

## The House of Vision Inc.

CHICAGO

EVANSTON
DES MOINES
HIGHLAND PARK
MASON CITY
MUSKEGON
OAK PARK
MILWAUKEE
SIOUX CITY
AURORA
MINNEAPOLIS

### THE VALUE OF GRINDING MINUS CYLINDERS IN STRONG ASTIGMATIC CORRECTIONS

The House of Vision has had a standing rule for the past twenty-five years that all astigmatic corrections over 2.00 diopters must be transposed to a minus cylinder and ground on the concave surface of the lens wherever possible. This rule at first was the result of the observation that persons wearing these strong cylinder corrections were much more comfortable with this form of grinding. In addition, the lenses were better from a cosmetic standpoint.

The scientific reasoning back of this rule is that correcting refractive error at a distance from the eye changes the size of retinal images in a way that increases with that distance and with the correction. Astigmatic lenses cause different changes in different meridians, so that squares look like diamonds, and verticals can lean by nearly a degree for every diopter of cylinder,—enough to disturb balance in a sensitive patient. That's not all. When a nearby object is made to look larger or smaller than normal, the light seems to come from a point nearer or farther than the object, and requires accommodation different from normal. In this case, the effect is different in meridians of different power, so that additional astigmatic correction is theoretically required for near vision, nearly one-fifth of that prescribed for distance. Of course, grinding a bifocal lens with a stronger astigmatic correction in the bifocal only, presents grinding complications. In certain cases it is almost impossible.

The disturbing effects described above are reduced by the practice of grinding strong astigmatic corrections on the concave surface of the lens and fitting those lenses as close to the patient's eye as possible. This helps the patient to accept such corrections even when worn at oblique axis or for the first time.

Practical experience, scientific research and exacting craftsmanship join in presenting another contribution to clear and comfortable vision.

*"if it's a lens problem, let's look at it together"*

THE HOUSE OF VISION—MAKERS OF PRESCRIPTION GLASSES FOR THE MEDICAL PROFESSION—WILL BE HAPPY TO FILL *YOUR* PRESCRIPTIONS.

AMERICAN JOURNAL OF OPHTHALMOLOGY

SERIES 3 · VOLUME 38 · NUMBER 1 · PART I · JULY, 1954

# CONTENTS

ORIGINAL ARTICLES

Endocrine exophthalmos: An evaluation of present knowledge. The Fourth Estelle Doheny Eye Lecture. Frederick C. Cordes ..... 1

A new trephining operation. Frederick H. Verhoeff and Paul A. Chandler ..... 21

Argyll Robertson pupil: Following herpes zoster ophthalmicus: With remarks on the efferent pupillary pathways. Howard A. Naquin ..... 23

The significance of the unilateral Argyll Robertson pupil: Part I. A report of 13 cases. Julia T. Apter ..... 34

Bilateral homonymous hemianopsia. Frederic M. Reese ..... 44

Exotropia with bilateral elevation in adduction: Part I. Diagnosis. Martin J. Urist ..... 58

Postnatal cataracts in premature infants. Loren P. Guy ..... 65

An apparatus for investigation of the eye: In monochromatic light with interference filters. M. Hultin and T. Kornerup ..... 68

Loss of the lower fornix of the anophthalmic socket. Alston Callahan ..... 75

Retrobulbar injection of Lidocaine (Xylocaine®) for anesthesia and akinesia. Dan A. Russell, Jr., and Jack S. Guyton ..... 78

Use of parenteral trypsin in acute inflammatory and thrombotic ocular disturbances. Joseph M. Hopen ..... 84

Clinical pathologic conference. Parker Heath ..... 88

NOTES, CASES, INSTRUMENTS

Magnetic foreign body: Report of an intraocular case in which removal was successful after 24 years. Gregory Flynn and Morgan Raiford ..... 90

Orbital retractor. Robert L. Alexander and Peter H. Ballen ..... 91

Improved orbital implant. H. Arruga ..... 93

Office treatment tray. Richard J. Carbo, Jr. ..... 95

SOCIETY PROCEEDINGS

Yale University Clinical Conferences, March 13 and 27, 1953 ..... 97

Minneapolis Ophthalmological Society, May 4, 1953 ..... 100

Memphis Society of Ophthalmology and Otolaryngology, May 12, 1953 ..... 105

EDITORIAL

Diamox and the therapy of glaucoma ..... 109

CORRESPONDENCE

A Brigadier General ..... 111

BOOK REVIEWS

The Year Book of the Eye, Ear, Nose and Throat ..... 111

Textbook of Ophthalmology ..... 112

ABSTRACTS

Anatomy, embryology, and comparative ophthalmology; General pathology, bacteriology, immunology; Vegetative physiology, biochemistry, pharmacology, toxicology; Physiologic optics, refraction, color vision; Diagnosis and therapy ..... 114

NEWS ITEMS ..... 127

**A balanced combination of decongestant, astringent and antiseptic, OpH promptly soothes minor ocular irritations.**

**OpH formula:**
Neo-Synephrine® hydrochloride (0.08%)
zinc sulfate (0.06%)
boric acid (2.2%).

***OpH is sterile, buffered and isotonic with tears.***

***Supplied in new, handy individual dropper bottles (10 cc.) which prevent dropper contamination.***

**Specify-OpH-new sanitary ophthalmic preparation in prescription type package.**

**WINTHROP-STEARNS INC.** New York 18, N. Y. • Windsor, Ont.

Neo-Synephrine (brand of phenylephrine), trademark reg. U. S. & Canada

AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 38 JULY, 1954 NUMBER 1

# ENDOCRINE EXOPHTHALMOS: AN EVALUATION OF PRESENT KNOWLEDGE*

THE FOURTH ESTELLE DOHENY EYE LECTURE

FREDERICK C. CORDES, M.D.
*San Francisco, California*

The honor conferred upon me by the trustees of the Estelle Doheny Eye Foundation in asking me to present the fourth in this series of lectures is deeply appreciated.

Mrs. Edward Laurence Doheny is one of the great benefactresses of the State of California, and of Southern California in particular. Her interest in the history and welfare of the state that has been her home since childhood has been unflagging. California was also dear to her husband, and it was here that he drilled the first oil well from which stemmed the family fortunes.

Mrs. Doheny has made many generous contributions to her community. Most of them have been kept modestly anonymous, but I should like to take the liberty of mentioning two notable gifts she has made in the educational field: the Doheny Library on the campus of the University of Southern California and the Edward Laurence Doheny Memorial Library at St. John's Seminary in Camarillo. The latter houses one of the finest rare-book libraries in the United States and is an enduring contribution to the scholars and historians of present and future generations.

Mrs. Doheny's interest in creating a foundation dedicated to the conservation and restoration of vision resulted in the establishment of the Estelle Doheny Eye Foundation. In 1947, the foundation opened the Doheny Laboratory at St. Vincent's Hospital to provide pathologic, bacteriologic, and other ophthalmologic services for the community, and the Eye-Bank for Southern California was established in connection with it. As a further service to ophthalmology, the foundation recently made a grant to the University of California at Los Angeles for research on the vitreous body.

The original work that has been done at the Doheny Laboratory has already marked it as one of the more important ophthalmologic research laboratories in the country and has helped to signalize Los Angeles as an eye center of major importance on the Pacific Coast.

I know I express the sentiments of all the men in our specialty when I say that we are most grateful to Mrs. Doheny for her generosity in establishing this foundation. May it continue to grow in its service to mankind.

---

The extensive research that has been carried on in the field of endocrinology in the last 15 years, and especially in the last 10 years, has entirely changed our concept of the basis of endocrine exophthalmos and has altered radically our therapeutic approach to it. The results of numerous experimental and clinical studies, and the development of Naffziger's orbital decompression operation, have made it possible for us to save the vision of many patients who would formerly have become blind. In view of these advances, an evaluation of our present knowledge of the subject seemed to me to be in order.

* From the E. S. Heller Laboratories and the Department of Ophthalmology, School of Medicine, University of California, San Francisco. Presented in Los Angeles, California, November 5, 1953.

## Endocrine-gland physiology

In any consideration of exophthalmos of endocrine origin, it is essential to have some understanding of the general principles of endocrinology which relate to the ocular changes. These principles and relationships have been ably described by Williams.[1]

The evolution of man from a unicellular animal to a complex organism was accomplished by the introduction of the nervous system and the endocrine glands, each with its own channels, nerves, and blood vessels for the transmission of agents which influence body function. The nervous system produces effects rapidly, the endocrine glands relatively slowly. As integrators they initiate chains of reaction and help to produce a balance among the various forces involved. One of the chief goals of the integrators is to supply eneregy for heat, locomotion, mentation, and reproduction.

The central nervous system is frequently responsible for significant changes in metabolism; it may affect pituitary function, which in turn affects the body cells directly or indirectly through the so-called "target glands," or it may in some instances stimulate the target glands directly. Other reactions occur without the aid of the nervous system and are traceable instead to the influence of certain hormones or metabolic products on the pituitary gland, the target glands, or the general body cells themselves.

The afferent impulses of the neuroendocrine reflexes may be carried to the brain by the nervous system or may be psychogenic. The neuroendocrine relationship may be changed by the presence of a lesion involving the nerve tracts to the pituitary gland. The efferent nervous signals to the pituitary, which influence the amount of secretion of one or more hormones, are carried chiefly through the hypophyseal stalk.

The pituitary manufactures a wide variety of hormones; these include the growth hormone, the anti-insulin hormone, the adrenotropic hormone, and a number of other hormones that exert local and general effects. Glands directly affected by pituitary hormones have been referred to as target glands; for example, the thyroid gland, the adrenal gland, and the gonads, and their reaction-patterns may be regarded as laws of endocrinology. The pituitary hormones that stimulate them are designated by the name of the gland, plus the suffix -tropic or -trophic.

*Hypotropism.* When the production of a tropic hormone ceases, the related target gland atrophies and there is an associated decrease in the rate of the manufacture and release of its hormone into the circulation.

*Hypertropism.* Hypersecretion of a tropic hormone leads to hypertrophy and hyperplasia of the target gland, with an increase in the rate of manufacture and transfer of the target-gland hormone into the blood stream, and a decrease in the concentration of the hormone in the gland. When the amount of hormone produced by the target gland is distinctly less than is required by the body, there is an increase in the production of the tropic hormone, which leads to hyperplasia and hypertrophy of the target gland. This is the mechanism of the hypertrophies of the thyroid associated with iodine deficiency and with puberty and pregnancy.

According to William,[1] the mechanism of informing a gland as to the needs for its products varies, but the concentration of a specific hormone in the blood often appears to be the chief determinant.

## The thyrotropic hormone

The pituitary hormone with which we are concerned in this discussion acts in two ways: (1) To stimulate the thyroid gland, and (2) to induce systemic effects, notably on orbital structures. Thyroxin, the thyroid hormone, antagonizes both these effects; iodine depresses the first action only. When no thyrotropin is produced, as after hypophysectomy, the thyroid gland atrophies and the symptoms of athyreosis ultimately de-

velop. If thyrotropin is administered before the atrophy is pronounced, however, a normal physiologic and anatomic status can be temporarily restored to the thyroid. When the thyrotropin is given in large doses, moreover, a clinical picture closely resembling Graves' disease can be produced.

Response to thyrotropin is depressed by the simultaneous administration of potassium iodide or thyroxin. The mechanism of this thyroxin effect is not understood, but the potassium iodide is apparently converted to iodine by the thyroid, and in this form can inactivate thyrotropin by oxidizing it. It has been shown, however, that potassium iodide given to a normal individual does not completely inactivate thyrotropin since in this case the metabolic rate remains within normal limits.

The administration of thyrotropin promotes water retention, alters fat metabolism, and can produce a marked exophthalmos. The water content of the orbital structures increases and the extraocular muscles become markedly swollen and hypotonic; ultimately they show microscopic changes. The fat content of the orbit greatly increases and the skeletal muscles in general tend to lose some of their striations and to exhibit many tiny fat droplets. Fat vacuoles appear in the liver, kidney, and various epithelial cells, and are found in phagocytic cells of the lungs, spleen, and lymph nodes. Plasma fat and blood acetone are increased and the polymorphonuclear neutrophils may increase in the blood and their cytoplasms may exhibit many fat droplets.

### Physiology of the thyroid gland

Biochemically, the main function of the thyroid seems to be to produce a colloidal material containing a hormone which will stimulate the rate of metabolism in proportion to the demands of the body. Of the several substances comprising the colloid, the most important is thyroglobulin which contains iodine bound to certain of its amino acids to form the diiodotyrosine and thyroxin. Thyroxin, or tetraiodothyronine, is either the thyroid hormone or its main constituent and is found in a larger concentration in thyroglobulin than elsewhere.

The thyroid hormone is formed, presumably, in the cells of the thyroid gland, but its iodination could occur both in the cells and in the lumina. It has been shown that a compound with biologic properties similar to those of the thyroid hormone can be formed in the total absence of cells:

Lerman and Salter[2] found that the incubation of serum albumin with elemental iodine in vitro, without the addition of any enzymes, would produce such a compound, and Reineke and Turner[3] observed that the iodination of mild proteins could produce a compound with marked thyroidal activity. Williams states, however, that in spite of the apparent ease with which thyroxin can be manufactured in nonthyroidal sites, the thyroid gland is still necessary because of its capacity to produce the hormone much more rapidly than other tissues or fluids and because it stores in its follicles an extra supply which can be mobilized rapidly.

The main functions of the thyroid hormone are to stimulate the metabolism, growth, development, and differentiation of tissues. It increases the rate of energy exchange, as indicated by increased oxygen consumption, and promotes an increase in the rate of growth of essentially all the tissues of the body. It decreases the fatty acids, cholesterol, and phospholipids of the serum, and increases the demands for vitamins, minerals, and water.

Williams[1] has summarized the more important aspects of the physiology of the thyroid gland as follows:

1. The stimulus for increased thyroid function is apparently set off by a deficiency in thyroxin. The pituitary is stimulated, presumably through the hypothalamus, to an increased production of thyrotropin; this causes (a) a more rapid transfer of thyroid

hormone into the blood stream, (b) hypertrophy and hyperplasia of the acinar cells, (c) an increase in thyroid iodide, (d) increased oxidation of iodide to iodine in the thyroid, (e) an increase in the rate of synthesis of diiodotyrosine and thyroxin, and (f) decreased storage of thyroid hormone.

2. A decreased production of the thyroid hormone occurs when there is a deficiency in the body supply of iodine or when, in the presence of a normal supply of iodine, its synthesis is antagonized by one of the thioureas.

3. With marked and prolonged stimulation, a varied response may occur in the acini. There may be varying degrees of hypertrophy and hypotrophy and variations in the colloid to organic iodine ratios.

4. Thyrotropin may be inactivated by thyroid tissue or nascent iodine, and reactivated by thiouracil and other reducing agents.

5. The exact nature of the thyroid hormone and its specific mechanism of action are unknown.

6. There is an increase of a thyroxinlike substance in the blood and muscles of patients with hyperthyroidism, and a decrease in those with hypothyroidism.

Under normal conditions there is a state of equilibrium between the endocrine glands, each producing and secreting sufficient quantities of its hormone to meet the systemic needs. Thus, when the thyroxin output drops beneath the body need, the pituitary gland secretes thyrotropin to stimulate the thyroid gland to greater activity. When the thyroid activity is increased, there is a decrease in the output of thyrotropin. It is the disturbance of this balance over a long period of time that produces the exophthalmos of endocrine origin with which we are here concerned.

## Physiology of the Exophthalmos Associated with Thyroid Dysfunction

The association of exophthalmos with thyroid disease was first observed many years ago. Credit for the discovery is often given to Basedow (1840),[4] although it has been established that the association was described by Parry[5] in 1776 and by Graves in 1835.[6] In Osler's opinion,[5] the account left by Parry was so complete that it more justly entitled him to the honor of having been the first to identify exophthalmic goiter, but Zondek[7] has pointed out that Basedow was the first to describe the exophthalmos as related to, and caused by, the goiter.

There has been a good deal of speculation in the past as to the cause of the exophthalmos associated with thyroid disease. In a review of the literature, Naffziger[8] found that the following causes had been suggested: (1) Increase of retrobulbar fat; (2) sympathetic stimulation of the smooth musculature of the orbit; (3) abnormalities of Müller's muscle; (4) stasis of the lymph vessels of the orbit; (5) retrobulbar venous engorgement; (6) varicosities of the orbital veins; (7) dilated orbital arteries, and (8) weakening and elongation of the ocular muscles.

Tilley[9] attributed this type of exophthalmos to the fact that the wide open lids exerted no restraint on the globe and thus permitted an increase in the orbital contents. None of these theories has satisfactorily explained all cases.

In 1920, Moore[10] reported the case of a woman who had severe ocular proptosis which resisted lid sutures and from which he removed a teaspoonful of edematous fat. This procedure made possible the approximation of the lids. He observed that in this particular case the exophthalmos was due to retrobulbar pressure and concluded that this process, or some modification of it, was probably the cause of the exophthalmos in Graves' disease.

The histologic examination of a portion of the inferior oblique muscle removed in the course of the procedure revealed pathologic changes similar to those subsequently described by Burch,[11] Naffziger,[8] and others.

In 1922, Moore[12] stated that the immediate cause of the exophthalmos in Graves' disease was an increase of orbital fat, perhaps combined with edema of the tissues, and that there was no satisfactory evidence of any other cause. At about the same time there appeared in the literature the first reports of cases of progressive exophthalmos that had apparently been induced or aggravated by thyroidectomies for the alleviation of Graves' disease.

In 1944, Mulvany[13] presented a new concept of the anomaly. He classified the various forms of hyperthyroidism occurring spontaneously in man in three categories: thyrotropic, thyrotoxic, and thyropathic.

The first, depending on pituitary control, is familiar as a physiologic and pathologic development in the latter third of pregnancy and in acromegaly.

The second is exemplified in exophthalmic goiter, a compound expression of hyperthyroidism and sympatheticotonia which Mulvany felt was maintained independently of thyrotropic activity.

The third, arising from local diseases of the thyroid gland, is often seen in early phases of neoplasm, in infections, and in lymphadenoid goiter.

Mulvany[14] stated that thyroid disease in only two of these categories appeared generally to be associated with exophthalmos; that is, (1) thyrotoxicosis proper and (2) thyroid disease of thyrotropic origin. He felt that these were two distinct diseases, one the result of thyrotoxicosis independent of hypophyseal control, and the other of pituitary origin. He was the first to point out that there were differences in the clinical findings and courses of these two forms of exophthalmos.

Experimental exophthalmos has been produced by a number of substances. Sellers and Ferguson[15] found that rats fed propylthiouracil for seven months developed exophthalmos. It disappeared during sleep, however, and showed no signs of inflammation, edema, or increased orbital fat.

Loeb and Friedman[16] and Marine and Rosen[17] showed that hyperthyroidism with exophthalmos could be produced by the injection of pituitary extract containing thyrotropic hormone.

Probably the most important work in this field has been done by Smelser,[18–23] who demonstrated that the easiest way to produce typical exophthalmos in guinea pigs was to remove the thyroid gland and, after recovery, to give the animal anterior pituitary extract; a certain number developed exophthalmos spontaneously without the addition of the anterior pituitary hormone. An exophthalmos could also be produced by the injection of large amounts of thyroxin. The response of the animals varied greatly, but all those that had been previously thyroidectomized promptly developed an exophthalmos; its degree could be reduced by the use of both iodine and thyroxin, and, since the animals under experimentation were hypothyroid animals, these results seemed to indicate that the use of thyroid extract would act as a barrier against the further release of thyrotropic hormone.

Similar views were expressed by Marine[20] who claimed that thyroid deficiency was one of two essential factors underlying the development of exophthalmos in Graves' disease, and by Brain[27] who stated that in exophthalmic goiter the exophthalmos was not the result of the hyperthyroidism.

Marine[26] made another interesting observation. He noted that a greater degree of exophthalmos appeared in those animals which were most active sexually, and concluded that the degree of functional activity of the interstitial cells, and possibly of the adrenal cortex, was an important factor in the development of exophthalmos.

Mulvany[13] felt that these conclusions offered for the first time an explanation of the absence of exophthalmos in other varie-

ties of thyrotropic hyperthyroidism.

Smelser and Ozanics,[28] on the other hand concluded from their work that the experimental type of exophthalmos was not affected by the absence of gonads or by an excess of androgens. Gonadectomy, or testosterone administration, did not affect the protrusion of the eye or the mass of orbital tissue in the guinea pig, and injection of adrenal cortical hormone had no effect on the orbital tissue of either control or exophthalmic animals. Gonadectomy, in the female, did not influence the exophthalmos, but the administration of large amounts of estrogen reduced it in 10 of 11 animals. Smelser and Ozanics regarded this finding as consistent with the generally higher incidence of progressive exophthalmos in males and with its greater incidence in women in the menopausal age group than in younger women.

In another series of guinea-pig experiments, these investigators[29] found that animals in which exophthalmos had been induced by thyroidectomy and injection of pituitary extract developed edema of the orbital fat and to a lesser extent edema of the fat in other areas of the body. Fat in some areas of the body showed no change, however. If some of this unchanged fat was transplanted to the orbit of a guinea pig and exophthalmos was then produced, the transplanted fat did not develop edema. However, if orbital fat from one guinea pig was transplanted to the orbit of another guinea pig, this fat developed edema when exophthalmos was produced. From this it was concluded that the reaction of the orbital fat depended on its nature rather than on its location.

Jefferies[30] injected various doses of pituitary extract into guinea pigs and found no differences, either in the exophthalmos or in the fat deposition in the livers, between intact and thyroidectomized animals.

This author[31] studied further the effects of iodination of pituitary extract in relation to the thyrotropic exophthalmic, and fat-mobilizing principles. He found it possible to inhibit almost completely the thyrotropic activity of the extract on the thyroid gland of guinea pigs while maintaining considerable exophthalmic and fat-mobilizing activity, and concluded that the thyrotropic hormone might not be the chief or only factor in the pituitary which could produce exophthalmos and fat mobilization in the liver. The treatment of the iodinated extract with thiouracil partly restored its thyrotropic activity and slightly increased the exophthalmos and fat-mobilizing effects.

Ludwig, Boas, and Soffen[32] observed that the development of experimental exophthalmos in the guinea pig was dependent on the accumulation of large quantities of intercellular ground substance and water in the connective tissue of the orbit. They found that there was an increase of hyaluronic acid, hexosamine, and water in these tissues. This is similar to the changes observed in localized myxedema in man and suggests a common mechanism for the two conditions.

Asboe-Hansen and Iversen[33] found that the subcutaneous injection of large doses of thyrotropin into guinea pigs resulted in a general fat mobilization and an accumulation of fat in the liver, muscles, and kidneys within 24 hours. There was a collection of mast cells and of a mucopolysaccharide, probably hyaluronic acid, in the connective tissue at the various sites, particularly in the retrobulbar tissue. They felt that the capacity of hyaluronic acid to bind water might possibly explain the edema and proptosis.

The exophthalmos-producing and liver-fat mobilizing effects of administering ACTH and 11-desoxycorticosterone (DOCA) to guinea pigs was studied by Jefferies.[34] He found no exophthalmos and no significant liver-fat deposition. Dayton[35] also found experimental exophthalmos in guinea pigs unaffected by ACTH, but in Aterman's[36] experience the condition was worsened by both cortisone and ACTH.

The weight of experimental evidence to date would seem to indicate that the effect

of the thyrotropic hormone is at least partially responsible for the production of the exophthalmos under consideration.

### Clinical definition of endocrine exophthalmos

According to Mulvany's[13] classification, there are two clinical types of endocrine exophthalmos, "thyrotoxic" and "thyrotropic." That the two types have a common basis, however, has been attested by Means, Hertz, and Williams,[37] Mann, [38] and others, and the underlying mechanism has been ably described by Martens[39]: Thyroid-stimulating hormone from the pituitary causes exophthalmos and also stimulates the thyroid gland to produce thyroxin. The thyroxin in turn inhibits the formation of thyroid-stimulating hormone by the pituitary.

In so-called "thyrotoxic exophthalmos," there is an excess of thyroid-stimulating hormone produced by the pituitary, possibly as a result of nervous stimulation through the hypothalamus. The excess thyroid-stimulating hormone causes the normal thyroid to respond by hypertrophy and an excessive secretion of thyroid hormone, and also, in some 50 percent of cases, initiates an exophthalmos. There is also a sympathetic hypertonia, probably caused by thyrotoxin directly or by associated changes in the adrenals. The excessive thyroid hormone inhibits thyroid-stimulating hormone formation and this prevents the exophthalmos from progressing.

In the "thyrotropic exophthalmos," there is initially the same excessive production of thyroid-stimulating hormone, but for some unknown reason the thyroid gland is unable to respond normally with the production of large amounts of thyroid hormone. Uninhibited by thyroid hormone, the pituitary continues to secrete still more thyroid-stimulating hormone and the exophthalmos progresses. The thyroid gland becomes hyperplastic but does not increase its output of thyroid hormone. In such a case the exophthalmos continues to increase and at a certain point the pathologic process becomes irreversible.

This was the case with the thyroidectomized guinea pigs already mentioned. In addition, as Braley[40] has pointed out, there may be an increase in the production of thyroxin without a change in the secretion of the anterior pituitary, in which case one may expect hyperthyroidism without exophthalmos; and there may also be a marked increase in the secretion of the anterior pituitary hormone without an appreciable increase in thyroid gland secretion; in this event, the exophthalmos is associated with a normal metabolic rate.

For the purposes of the ensuing discussion, a somewhat different classification will be employed. In broad terms, the exophthalmos under consideration will be considered as *progressive* or *nonprogressive*, since in my opinion the ocular changes that occur in thyrotoxicosis, and which include a mild, nonprogressive exophthalmos in some 50 percent of cases only, should not be confused with the progressive disease ordinarily referred to as "thyrotropic exophthalmos" in which, except in rare instances, an excess of thyroid-stimulating hormone is always to be found circulating in the blood.

### I. The nonprogressive exophthalmos of thyrotoxicosis

Thyrotoxicosis occurs most commonly in young adults, and women are more often affected than men. The typical case presents an elevated metabolic rate, weight loss, tachycardia, and tremor. The ocular manifestations include lid signs, eye signs, extraocular muscle changes, and nonprogressive exophthalmos in 50 percent of cases.

Many lid signs have been described. Widening of the palpebral fissure (Dalrymple's sign) is due to lid retraction and results in a staring expression; according to Soffer,[41] it is noted in about 18 percent of patients with diffuse goiter. Lid-lag (von Graefe's sign) is much more frequent, oc-

curring in four of every five patients with toxic goiter. Other signs include infrequent blinking (Stellwag's sign), absence of gaze (Jeffery), resistance to eversion of the lid (Gifford), and tremor of the closed lids (Rosenbach). The mechanism of the production of these signs is unknown; they may be of central nervous origin or may be due to instability of autonomic innervation.

The extraocular changes include weakness of convergence (Moebius), pigmentation of the skin of the lids (Jellinek), and dilatation of the pupil following the installation of adrenalin into the conjunctival sac (Loewi). The reason for these signs is also obscure.

Myasthenia is a constant accompaniment of exophthalmic goiter. The eye muscles are affected to a greater extent than other skeletal muscles, and ocular palsies of individual muscles may occur. According to Mulvany,[14] ptosis and interference with lateral movement are the most common myasthenic effects. Lyle[42] states that in muscle paresis there is usually contracture of the direct antagonist rather than of the yoke muscle. He feels that the most common paresis is of the superior rectus, with inferior rectus fibrosis and contracture. Moore[43] says that although paralysis of some of the external ocular muscles may occur, ophthalmoplegia interna does not.

According to Woods,[44] exophthalmos develops in over half the patients with diffuse toxic goiter. The degree may be measured with the exophthalmometer.

The average normal measurement with the Hertel exophthalmometer ranges from 11 to 20 mm., and in some patients with thyrotoxicosis there may be an increase of from 1.0 to 3.0 mm.; the increase is usually not more than 1.5 mm., and the exophthalmos is never progressive. Often, indeed, it is more apparent than real, because of the widening of the palpebral aperture by retraction of the lids.

There is no evidence of increased retrobulbar pressure; moreover, by firm and gentle pressure it is possible to push the globe back into the orbit. Congestive features are also absent, lid edema and chemosis never occurring except as a result of local infection. The exophthalmos is rarely unilateral or asymmetric. The vision is normal, there is no change in the visual fields, the intraocular pressure is normal, and the signs associated with the thyrotoxicosis disappear after thyroidectomy (Dobyns,[45]).

### 2. Progressive exophthalmos

Progressive (thyrotropic) exophthalmos develops during the course of a mild hyperthyroidism, following thyroidectomy, or spontaneously without a history of thyrotoxicosis. The condition has been described under a variety of titles which include exophthalmic ophthalmoplegia, malignant exophthalmos, progessive exophthalmos following thyroidectomy, and hyperophthalmopathic Graves' disease. It affects middle-aged adults primarily and is more common in men than in women. According to Martens,[39] the urine shows an excess of thyroid-stimulating hormone.

Poppen[46] found that patients with toxic exophthalmos showed persistence of the exophthalmos after thyroidectomy in 21 percent of cases, but an increase of the exophthalmos in only 1.1 percent. The time of appearance of the progressive exophthalmos varied from two weeks to 25 years after the thyroid surgery.

The signs of progressive (thyrotropic) exophthalmos include true proptosis, photophobia, lacrimation, foreign-body sensation, bulging of the lids, retrobulbar resistance, extraocular muscle weakness, and diplopia. It is more likely to be unilateral than the nonprogressive type and the bilateral cases are more often asymmetric.

The proptosis is always real (never spurious, as it often is in thyrotoxicosis), uncomfortable, and accompanied by the symptoms already described. Lid spasm is absent so that the lids can be everted easily in the

early stages. Definite retrobular resistance prevents the globe from being pushed back easily into the orbit.

Early in the disease conjunctival edema and congestion make their appearance and progress in severe cases until corneal ulceration ensues from pressure and exposure.

As pointed out by Mulvany,[14] the lacrimal gland is constantly affected by changes similar to those observed in the extraocular muscles. Naffziger[8] noted that in four of his cases the lacrimal glands were enlarged and readily palpable.

Eye movements are affected early according to Mulvany,[14] partly because of mechanical hindrance and partly as a result of muscle degeneration. The levator and orbicularis are usually spared. Upward movement of the eyes is disturbed most frequently, lateral movement less often, and depression least of all. Total immobility is not uncommon. The weakness of the ocular movements is not altered by prostigmine as it is in nonprogressive exophthalmos, and the limitation of motion often becomes worse after thyroidectomy.

From a clinical standpoint Mann[35] divided these cases into the following three groups:

1. Progressive exophthalmos without preceding thyroid disturbance; that is, primary thyroxin deficiency with compensatory excess of thyrotropic hormone. These cases respond well to thyroid extract and tarsorrhaphy.

2. Progressive exophthalmos with a history of previously treated hyperthyroidism and subsequent excess of thyrotropic hormone. These are cases in which there has been an excess of thyroxin followed by thyroid failure or thyroidectomy with subsequent excess of thyrotropic hormone.

3. Progressive exophthalmos in which an excess of thyroxin and an excess of thyrotropic hormone arise simultaneously.

*Complications of progressive exophthalmos.* Localized areas of myxedema of the extremities may be associated with thyrotropic exophthalmos. Miles and Forsey[47] found myxedema present in three percent of postthyroidectomy cases. Grynkewich and his co-workers[48] found the myxedema most commonly on the anterolateral surface of the lower leg. It may be an indication of future progression of the exophthalmos and should therefore be regarded as a warning signal, but it may also regress spontaneously. A clear relationship between thyrotropic exophthalmos and myxedema was also observed by Scheie and Hedges.[49]

Vail[50] found an associated myasthenia gravis and suggested a prostigmine test for every patient with thyrotropic exophthalmos. He also observed that some patients with thyrotropic exophthalmos showed acromegalic facies.

Beierwalters[51] states that glaucoma is sometimes associated with malignant exophthalmos and that it is usually improved by control of the exophthalmos. Givner and his co-workers[52] found an occasional rise of ocular tension because of pressure of the upper lid on the globe; they found no field loss, however, and felt that treatment of the glaucoma was unnecessary.

Braley[53] made some interesting observations on the intraocular pressure in endocrine exophthalmos. He found that in the nonprogressive (thyrotoxic) form, the intraocular pressure was never high, was in fact apt to be low (around 15 mm. Hg, Schiøtz), and did not change with change of the position of the head. In the progressive (thyrotropic) type, the tension was usually normal if the eyes were in the primary position of rest; if, however, with the patient on the table, the chin was elevated or depressed and the eyes fixed straight up, the tension rose.

Although Brown and Schwartz[54] reported an associated optic neuritis in one case of nonprogressive, thyrotropic exophthalmos, it is generally believed that there are no fundus changes in this form of the disease. In progressive (thyrotropic) exophthalmos, how-

ever, Walsh[55] records observing papilledema, neuroretinal edema, and dilatation and tortuosity of the retinal vessels.

Dobyns[45] found that severe cases sometimes showed papilledema or engorgement of the retinal veins, and Mulvany[14] reported disc changes with resultant optic atrophy.

In one patient which I observed personally, there were folds in the retina around the optic nerve and subsequent partial loss of vision and partial optic atrophy.

In four of his six cases, Naffziger[8] found changes in the optic nerves and retinas, especially swelling of the nervehead, hemorrhages, and atrophy. Mann[56] states that the intraocular signs include papilledema, retinal venous congestion, and partial optic atrophy.

Nonspecific field changes and marked diminution of visual acuity may occur in the more severe cases of progressive exophthalmos. Naffziger[8] reported generalized contraction of the fields in two cases and loss of the inferior half of the field in another.

In one case observed by me, the loss of field was such that the right eye retained only a crescent in the 30 to 40-degree arc superiorly; the left eye showed a mild general contraction.

Mulvany[14] also regarded general contraction of the fields due to disc changes as likely to occur in these cases. Ernst Fuchs[57] explains these changes in the optic nerve as a result of undue stretching. If this stretching takes place gradually, the nerve fibers often accommodate themselves to it so that they preserve their conductivity, and the vision is maintained intact; but if the exophthalmos increases rapidly, the traction on the optic nerve soon deprives it of its conducting power.

Mann[56] expressed the opinion that the intraocular signs and field defects could all be explained as pressure and traction effects.

*Effect of pregnancy.* The behavior of thyroid disorders during pregnancy is noteworthy.

Givner, Bruger, and Lowenstein[52] reported a case of a 34-year-old white woman whose exophthalmos had increased following thyroidectomy but had finally stabilized. She subsequently became pregnant and at the beginning of the second trimester the exophthalmos showed improvement which continued up to the time of delivery. Exophthalmometer readings showed a recession of four mm. The baby was born with a severe exophthalmos which did not recede until the child was a year old. At the end of three years there was no evidence of exophthalmos in the child and the mother's exophthalmos had remained at the new low level.

Another interesting case was reported by Frisk and Josefsson.[58] A woman with thyrotoxicosis treated with methylthiouracil became pregnant after 14 months of therapy. The treatment was continued throughout pregnancy. At birth the child showed no abnormalities and the thyroid gland was not enlarged. In the second month of life a marked change in the temperament of the baby occurred simultaneously with the development of exophthalmos and a drop in blood cholesterol. The symptoms of excitability and exophthalmos decreased over the next seven months. The authors explained this as probably a result of the passage of thiouracil through the placenta with consequent blockage of the fetal synthesis of thyroid hormone and increased activity of the fetal hypophysis. The thyroxin produced by the mother prevented myxedema of the fetus and helped suppress excessive thyrotropic activity. After birth and withdrawal of the thiouracil, the hyperfunction of the pituitary persisted for some time and gave rise to the exophthalmos and other symptoms.

*Effect of psychic trauma.* Brain,[27] Mann,[56] and others have observed that patients with endocrine exophthalmos frequently give a history of shock or worry.

Givner and his associates[52] state that an increase in the exophthalmos of hyperthyroidism may be related in part to such factors as security, frustration, violent emo-

tional trauma, and infection. In one of their cases the exophthalmos increased rapidly following the mother's agitation when her son, a navigator in the air force, was sent overseas; within six weeks her exophthalmos increased 2.5 mm. in one eye and 3.0 mm. in the other.

Another example was that of a 29-year-old woman who took thyroid extract to induce loss of weight. She developed hyperthyroidism and acquired a stare. A thyroidectomy was performed and soon thereafter the exophthalmos began to progress slowly. Nine months after operation her father became acutely ill and she attended him constantly. She then noted a rapid progression of the exophthalmos which reached 30 mm. in the right eye and 31 mm. in the left.

### 3. Hypophyseal exophthalmos without thyroid dysfunction

In his discussion of a case of unilateral hypophyseal exophthalmos, Iglesias[59] offers some interesting observations. In many cases of exophthalmos due to cerebral tumors, the lesions are hypophyseal tumors. If the exophthalmos appears as the only symptom, without any hyperthyroid manifestations, it is difficult to conclude that it is due to the thyrotropic hormone since this hormone invariably produces, in addition to the exophthalmos, more or less intense hyperthyroid manifestations. It is therefore possible, as has already been suggested, that the exophthalmos is due to a hormone distinct from the thyrotropic hormone, an "exophthalmos-causing" hormone. Among the cases of purely hypophyseal exophthalmos, the commonest cause is a chromophobe adenoma.

The case of progressive exophthalmos described by Medine[60] provides further evidence of the possibility of a purely hypophyseal type of exophthalmos. In his patient it was not possible to show any abnormality of thyroid function, nor was it possible to demonstrate any disturbance in the relationship between the thyrotropic hormone and the thyroid gland. He felt justified in concluding that in this case an excessive production of the pituitary factor was responsible for the exophthalmos without a clinically significant increase in the thyrotropic factor.

In 1934, Dunnington and Berke[61] described four cases of exophthalmos due to chronic orbital myositis. They were characterized by painless edema of the lids of gradual or sudden onset, moderate to severe exophthalmos, limitation of ocular movement, and inelasticity of the ocular muscles. Histologic examination revealed lymphocytic infiltration, fibrosis, and degeneration of muscle fibers. Microscopically, the picture could not be differentiated from that of the progressive exophthalmos associated with thyroid disease. There was no evidence of thyroid disease, however, and no demonstrable cause for the condition. It is interesting to speculate that these cases may also have been the result of a special "exophthalmos-causing" hormone.

### Asymmetric and unilateral exophthalmos

Endocrine exophthalmos may be asymmetric or unilateral. At the Mayo Clinic, Drescher and Benedict[62] found that 25 percent of the cases of asymmetric exophthalmos were of endocrine origin and that the asymmetry in cases of endocrine origin rarely exceeded 7.0 mm. Selinger[63] stated that unilateral exophthalmos occurred in some 10 to 20 percent of cases of thyrotoxic goiter, but Rundle,[64] Bedrossian,[65] and others have reported the exophthalmos of thyrotoxicosis as only occasionally asymmetric or unilateral. The progressive type is more likely to be unilateral and the bilateral cases are more often asymmetric.

In the absence of other manifestations of endocrine disturbance, the diagnosis of unilateral cases or of those with marked asymmetry may be very difficult. According to Benedict,[66] the exophthalmos of exophthalmic goiter may develop as much as several

months after a subclinical episode of mild exophthalmic goiter which has produced no noticeable general symptoms.

Saltzman and Mellicker[67] reported a case of exophthalmos that preceded the appearance of thyrotoxic symptoms by 20 months. They were able to find eight similar cases in the literature and concluded that delays of this magnitude occur more frequently than reported.

A case reported by Bedrossian[65] is in point. His patient, a 45-year-old man, had a right-sided exophthalmos that was diagnosed as an orbital tumor when the results of all studies were negative. The eye was enucleated and an examination of the orbital tissue showed an inflammatory process. A year later, the left eye showed similar changes but at this time a diagnosis of progressive (thyrotropic) exophthalmos could be made. A reexamination of the slide of the enucleated right eye revealed histopathologic findings consistent with thyrotropic exophthalmos.

## Pathology

The histopathology of endocrine exophthalmos has been subjected to considerable study with general agreement as to the characteristic findings.

### Pathology of nonprogressive exophthalmos

According to Mulvany,[14] the exophthalmos of thyrotoxicosis is caused by spastic contraction of Landstrom's and Müller's orbital muscles, combined with a so-called "myasthenia" of the extraocular muscles, and is characterized by normal orbital contents, absence of intraorbital edema, spastic retraction of the lids, and degeneration of the extraocular muscles of a kind sharply different from that found in the thyrotropic type.

Bedrossian[65] states that in the exophthalmos of thyrotoxicosis there is a general wasting of muscle fibers with loss of striation, fibrillation, and amorphous granulation of the sarcoplasm. The muscles are normal in size. There is no edema or round-cell infiltration but there is an increase in the amount of muscle and orbital fat. Fischer[68] reported a "true parenchymatous degeneration" in sections of recti muscles from eyes removed from cases of goiter.

### Pathology of progressive exophthalmos

Mulvany[14] attributes the thyrotropic type of exophthalmos to an increase of orbital contents with hypertrophy and degeneration of the orbital striated musculature. Bedrossian[65] describes the muscles as much larger than normal and as showing interstitial fibrosis, edema, and chronic inflammatory lymphocytic infiltration, often in perivascular form. In my own experience the examined muscles have been found to be markedly enlarged.

Gleason[69] reported an idiopathic myositis with muscles eight times the normal size. Falconer and Alexander[70] found edema of the interstitial tissue of the muscle, often associated with degenerative changes in the muscle fibers and with lymphocytic infiltration, focal or diffuse or both. They described a subsequent increase in fibrous tissue which replaced the degenerated muscle fibers and thickened the fibrous septa and sheaths.

In confirmation of the observations of other investigators, Falconer and Alexander[70] also found an excess of pituitary thyrotropic hormone circulating in the blood of patients with progressive (thyrotropic) exophthalmos. The hormone has also been found in the blood of most patients who have undergone thyroidectomies or thiouracil therapy for toxic goiter.

## Treatment

The treatment of endocrine exophthalmos varies according to the type of exophthalmos involved, whether nonprogressive or progressive, and will be discussed accordingly.

### Treatment of the nonprogressive exophthalmos of thyrotoxicosis

*Medical treatment.* The accepted modes of medical treatment include, according to Sof-

fer,[41] the administration of iodine, of the thiourea derivatives, and of radioactive iodine.

Although iodine was used in the treatment of Graves' disease as early as the 19th century, it was not until Plummer's [71] report in 1923 that it took its proper place in the treatment of the condition, particularly so far as the operative management of thyrotoxicosis was concerned. As has already been stated, the effect of iodine is to inactivate thyrotropin; its prolonged use may be associated with exacerbations of the disease.

The demonstration of Astwood[72] and others of the goitrogenic and hypothyroid-inducing potentialities of the thiourea derivatives was followed by the widespread use of these drugs in the treatment of thyrotoxicosis. The preparation most widely used is thiouracil. The major disadvantage of the thiourea derivatives is the relatively high incidence of the toxic reactions they induce. According to Soffer,[41] propylthiouracil is the most satisfactory of the presently available thiourea compounds, the incidence of toxic reactions from it being about two percent only. Agranulocytosis is uncommon but does occur.

Jones[73] found that applications of from 200 to 1,000 r to the orbits had some value in treating the exophthalmos of thyrotoxicosis, but X-ray treatment to the thyroid has been almost entirely abandoned as a therapeutic procedure in thyrotoxicosis since the introduction of radioiodine.

Radioactive iodine has proven to be so effective, indeed, that according to Soffer[41] it may in time replace the other forms of therapy in use today. Its effectiveness depends upon the capacity of the thyroid to concentrate iodine in its structure. According to Williams,[74] the most serious problem with radioiodotherapeusis is to select the appropriate dosage; there is only a narrow margin between an excessive dosage and a deficient dosage.

*Surgical treatment.* The results of the surgical treatment of hyperthyroidism are excellent in the properly prepared patient. According to Cattell,[75] a permanent cure is effected in 95 percent of cases. As Williams[74] points out, however, surgery causes death more often than do the other forms of treatment, and it can cause vocal cord paralysis and tetany; it necessitates hospitalization, moreover, and is more emotionally and physically upsetting to the patient than medical treatment.

*Effect of thyrotoxicosis therapy on associated exophthalmos.* The effect of the treatment of thyrotoxicosis on the associated exophthalmos has been reported by a number of observers. In over 60 percent of Dobyns'[76] patients treated with thiouracil, the exophthalmos increased more than one mm.; this was in contrast to the increase in only 10 percent of patients similarly treated by Beierwalters.[77] After X-ray treatment the exophthalmos in 20 percent of Soley's[78] patients progressed.

Soley, Miller, and Foreman[79] found a measured increase of the exophthalmos in 30 percent of the patients treated with radioactive iodine, in 26 percent of those treated with X rays, and in from 40 to 50 percent of those that had been subjected to subtotal thyroidectomy.

The rather general impression has been that exophthalmos improves after subtotal thyroidectomy. This impression of improvement is partly the result of the disappearance of lid retraction and other lid signs. Careful exophthalmometer readings, however, show that between 75 and 97 percent of patients show a postoperative increase in the ocular protrusion.[76, 78]

Givner and his associates[52] reported that ergotamine tartrate (1.0 mg., twice daily) reduced the stare with narrowing of the palpebral fissure but that the effects were transient only. Detroit[80] treated a patient with estrone and radiotherapy of the retro-ocular tissues; the exophthalmos regressed, but there was no change in the hyperthyroidism.

In the purely thyrotoxic type of case the

exophthalmos rarely becomes excessive so that an increase following therapy only occasionally poses a real problem. Cosmetic operations such as tarsorrhaphy may be performed in treated cases to improve the appearance.

### Treatment of progressive exophthalmos

Progressive (thyrotropic) exophthalmos is a much more serious therapeutic problem. As pointed out by Falconer and Alexander,[70] and by Dunnington and Berke,[61] the condition often appears to be self-limiting even though the regression may be incomplete. Because of this tendency, care must be exercised not to credit a therapeutic procedure in use at the time with what may be spontaneous improvement.

Thyroid therapy has been advocated for thyrotropic exophthalmos. Martens[39] reported fair success with thyroid hormone and ovarian hormone, and Craig[81] reported two cases of moderate exophthalmos and hypothyroidism treated by thyroid hormone and Lugol's solution with regression of the exophthalmos.

The majority of authorities feel that thyroid therapy is of little value in these cases, however. Thomas and Woods[82] reported failure with thyroid administration in five patients with progressive exophthalmos following thyroidectomy.

Falconer and Alexander[70] go so far as to say that in their experience thyroid therapy has neither prevented the development of the condition nor noticeably influenced its progress. One of their patients actually developed progressive exophthalmos after she had been taking thyroid regularly for more than two years to keep her weight down.

Mann[38] states emphatically that the cases she classifies as Group III (in which excessive thyroxin and excessive thyrotropic hormone arise simultaneously) cannot be given thyroid extract.

Thomas[83] reports that in one case of progressive exophthalmos, propylthiouracil seemed to slow the progress. In reports of single cases, however, one must always bear in mind the apparent self-limitation of some cases.

Stilbestrol and testosterone have apparently arrested or improved the disorder in some instances[39] but in Falconer and Alexander's series[70] they were of no benefit.

It has been reported that cortisone and ACTH may be beneficial. As has already been stated, however, Aterman[36] and others found experimental exophthalmos in guinea pigs worsened by these drugs, and in a symposium[42] on the management of endocrine exophthalmos, Aterman reported that cortisone and ACTH aggravated the condition.

One simple therapeutic program that should not be forgotten is limitation of fluids, elevation of the head of the bed, and quiet, all of which exert a favorable effect on orbital edema.

Experience has shown that the exophthalmos due primarily to excessive thyrotropin is frequently aggravated by thyroidectomy. Because of this, Soffer,[84] Martens,[39] Hermann,[85] and many others have stated that in the thyrotropic type of exophthalmos, subtotal thyroidectomy is contraindicated, and Falconer and Alexander[70] and others have come to feel that in patients with toxic goiter who also present some of the signs of excess thyroid-stimulating hormone, as opposed to mere lid retraction, it is wise to avoid thyroidectomy altogether.

Detroit[80] found that radiation therapy to the retro-ocular tissues caused regression of the exophthalmos, but no change in the hyperthyroidism. The results of irradiating the pituitary in these cases have varied.

Martens[39] reported poor results but Zonde and Ticho[86] described a case of progressive exophthalmos associated with myasthenia gravis in which the pituitary received 3,000 r with marked improvement in the myasthenia and the exophthalmos.

On the basis of 37 cases found in the literature, Dobyns[45] also says that X-ray

therapy of the pituitary is of value.

Beierwalters[51] gave X-ray therapy to 11 patients with progressive exophthalmos. He used 700 r through each of two temporal portals; the only side effects were transient epilation and headache. In eight of the patients there was a significant decrease (average 5.5 mm.) in the exophthalmos; the post-irradiation recession began at three months on the average, with the average maximum recession occurring at the end of 12 months.

Hermann[85] reported treating 12 cases of pituitary exophthalmos with radiation of the pituitary. With the "most rigid standard of assessment" she stated that seven of the 12 were improved, four dramatically and three moderately; she felt it could not be said with certainty, however, that the X-radiation was solely responsible for the improvement in the last three.

At the University of California, Low-Beer treated 11 patients with progressive exophthalmos with radiation to the pituitary. The treatment in no way affected the exophthalmos and no thyroidal endocrine effects were observed but the patients volunteered that they felt better; they no longer had headache, photophobia, or epiphora. All of them were observed for a period of six months after completion of the radiation.

Dr. Morris Daley, who is in charge of the thyroid clinic, stated that in his opinion the discrepancy between these results and some that have been reported is probably due to the fact that the University of California cases were of relatively long-standing exophthalmos with probably irreversible muscle changes.

Beierwalters[51] emphasizes that if exophthalmos has been present for 18 months, X-ray therapy is likely to fail.

### Surgery for ocular complications

Under certain conditions ocular surgery is indicated in endocrine exophthalmos to conserve vision and protect the cornea from exposure.

*Nonprogressive exophthalmos of thyrotoxicosis.* On some occasions there may be such excessive retraction of the upper lid in thyrotoxicosis that surgery is required for protection of the cornea. In this event the use of a lateral tarsorrhaphy is very effective. There may also be dislocation of the globe owing to the wide palpebral fissure and the laxity of the extraocular muscles. Tarsorrhaphy may be very helpful and may also be indicated for cosmetic improvement. Mulvany[14] states that tarsorrhaphy is useful in cases of corneal ulceration but that if there is sepsis the tension must be relieved.

*Progressive exophthalmos.* In a certain percentage of cases of progressive exophthalmos, the lids ultimately fail to cover the eye completely and the cornea is exposed. If not cared for, this invariably results in corneal ulceration, infection, and eventual blindness. A number of surgical procedures have been tried in these cases, including tarsorrhaphy, canthotomy, plastic operations on the conjunctiva, incisions of the lids, removal of orbital fat, the Krönlein operation, and Naffziger's complete removal of the orbital roof. More recently radical ethmoidectomy and a temporal decompression of the orbit have also been advocated.

Mann[39] is strongly in favor of lateral tarsorrhaphy. She treated a series of cases with endocrine products and sedative therapy, with and without tarsorrhaphy. No eyes were lost in 18 consecutive cases. Hermann[85] states that tarsorrhaphy alone cannot be expected to remedy rapidly raising intraorbital pressure. Whenever the eye cannot be closed and there is evidence of further progression of the exophthalmos, a combination of orbital decompression with tarsorrhaphy should be employed.

Mulvany[14] feels that tarsorrhaphy may aggravate the corneal damage and hasten loss of the eye. In his opinion immediate orbital decompression is required in these cases if the vision and the globe are to be conserved. Tarsorrhaphy would seem to have a definite

place, however, in severe cases, in which the progress of the exophthalmos has been arrested but in which there is difficulty in closing the lids.

The most generally used surgical procedure in these extreme cases is the Naffziger orbital decompression. The operative procedure consists of a complete removal of the orbital roof and lateral wall of the orbit by means of an intracranial approach. The orbital fascia is opened for purposes of decompression and to permit expansion of the contents of the orbit. Convalescence is characterized by a transient increase in the edema of the lids and conjunctiva. As it subsides the proptosis gradually lessens, and the recession of the globe, usually amounting to between 2.0 and 7.0 mm., continues over many months. The operation is followed by early improvement in vision, subsidence of papillitis, and disappearance of hemorrhages. The globe shows a faint pulsation of which the patient is unaware.

Poppen[46] reported a series of 66 cases of orbital decompression with no operative mortality. In his series the reduction of the exophthalmos ranged from 4.0 to 10 mm. after the decompression. Givner and his associates[32] stress the importance of uncapping the optic foramen if there is visual loss.

There are several reports in the literature of vision being helped by orbital decompression in the presence of papilledema, but Law[42] felt that in this circumstance the operation was a failure. It is at all events a major procedure carrying an operative risk so that it should be reserved for extreme cases.

Hermann[33] points out that the chief aim of orbital decompression is to relieve orbital edema and thereby to restore adequate drainage of the extraorbital tissues as well. She feels the operation should be reserved for patients who show rapidly increasing congestive changes with edema of the optic disc, or a visual defect from stretching of the optic nerve or retinal detachment.

In an attempt to evaluate the operation, Falconer and Alexander[70] record that in some cases it produces apparently striking results, that in others the eyeball does not recede, and that in still others, after a period of temporary recession, the eyeball protrudes once more as the pathologic process within the orbit advances. They were unable to foretell in individual cases the exact effect the operation would have. They conclude that in certain circumstances, as when ocular protrusion has been sufficient to produce corneal ulceration, the operation has proved a useful adjunct to treatment in spite of its limitations but that it is not the panacea which some authorities have claimed. They also suggest the more frequent use of lateral tarsorrhaphy as a palliative measure.

Medine[60] reports an interesting case in which the use of X ray to the orbits following orbital decompression reduced the exophthalmos.

Martens[59] states that radical external ethmoidectomy has had some value as a decompression operation for the orbit. It is a less formidable procedure, produces little scarring, and does not result in a pulsating globe.

Guyton[67] states that decompression into the subzygomatic or temporal fossa is as efficacious as intracranial decompression. He regards it as the least hazardous of the various decompression procedures since it is performed extracranially, in an aseptic field, and without danger to any important structures. He feels that, because of its benign nature, this operation can be recommended for patients desiring cosmetic improvement of exophthalmic disfigurement as well as for those threatened with impairment of visual function.

Naffziger,[68] however, feels that temporal decompression does not produce sufficient space for expansion to be effective in those cases in which it is performed to relieve the danger of visual loss.

One other procedure has a place in the surgery of endocrine exophthalmos. This is the modified Snellen suture for prolapsed

chemotic conjunctiva advocated by Laval and Schneider.[89] Occasionally after orbital decompression the redundant chemotic conjunctiva does not recede and the danger of corneal ulcer is increased. In such cases the following procedure is indicated:

After installation of pontocaine, cotton pledgets saturated with adrenalin 1:1,000 are applied to the prolapsed conjunctiva. This causes a reduction of chemosis so that the conjunctiva can be inverted with a muscle hook. A 3-0 black silk horizontal suture is inserted into the conjunctiva at the junction of the middle with the inner and outer thirds and 10 mm. from the lid margins. Each arm of the suture is then carried through the periosteum at the inferior orbital ridge and out through the skin of the cheek, and is tied over cotton. The sutures are removed on the 12th postoperative day. The procedure has the twofold purpose of restoring the prolapsed conjunctiva and of reforming the inferior fornix.

## Summary

The experimental work of many observers has shown rather conclusively that in exophthalmos of endocrine origin there is a disturbance in the balance between thyroxin (the thyroid hormone) and the thyrotropic hormone of the pituitary. The exophthalmos appears to be definitely associated with pituitary dysfunction and the proptosis occurs primarily in association with pituitary basophil excess. All of this work has changed earlier concepts of the condition and has necessitated the revision of many of our ideas on how to care for the affected eyes.

The clinical picture of endocrine exophthalmos has been well summarized by Mann.[90] A pseudoproptosis of the globe may be produced by lid retraction alone and in the early stages of thyrotoxicosis is very difficult to distinguish from the true proptosis which occurs when the pituitary thyrotropin level is high as well as the level of thyroxin. The degree of lid edema is helpful in determining the relative amounts of lid retraction and proptosis present. The proptosis occurs primarily in association with thyrotropic hormone excess and may be induced by hypothalamic stimuli, as in the progressive (thyrotropic) exophthalmos following fright or shock, or may be a part of a generalized anterior lobe dysfunction; the patient then shows signs of acromegaly and often of diabetes.

Progressive exophthalmos may complicate thyrotoxicosis or follow thyroidectomy. It may be associated with either a high or a low basal metabolic rate depending on the response of the thyroid to the increase in the amount of thyrotropin in the circulation. It may be accompanied by lid lag, lid retraction, or ptosis, as the thyroid response may determine. It may be unilateral and may or may not be accompanied by impaired ocular movement. In severe cases closure of the lids may be prevented and this may lead to corneal ulceration.

Although there are two clinical types of endocrine exophthalmos that have been designated as "thyrotoxic" and "thyrotropic," the present tendency is to regard them as fundamentally the same and different only in course and degree. The difference between them depends on the fact that in some cases of excessive thyroid-stimulating hormone there is an accompanying disturbance of the thyroid gland; in these cases there is almost always a demonstrable excess of thyroid-stimulating hormone circulating in the blood. For such cases it would seem appropriate to apply the term "progressive exophthalmos," reserving the term "nonprogressive exophthalmos," or perhaps "thyrotoxic exophthalmos" for those cases in which a mild exophthalmos results from an uncomplicated thyrotoxicosis; in these there is no excess of thyroid-stimulating hormone to be found circulating in the blood.

The presence of exophthalmos in cases of hypophyseal tumors in which no dysfunction of the relationship between thyroxin and thyrotropin can be demonstrated, and the rare case of exophthalmos in which there

appears to be no imbalance between the hormones, has led to the theory of an exophthalmos-producing hormone distinct from the thyrotropic hormone. In the vast majority of cases, however, it is possible to demonstrate an excess of pituitary thyrotropic hormone circulating in the blood of patients with thyrotropic exophthalmos and of patients after thyroidectomy or thiouracil therapy.

There is still considerable confusion regarding classification of types and nomenclature. For example, Falconer and Alexander were unable to make the histologic distinction between thyrotropic and thyrotoxic cases that was described by Mulvany. Their patients were all "advanced cases . . . referred to a neurosurgical unit because exposure keratitis had appeared or was deemed imminent." A review of the cases from which they had obtained material for microscopic study seems to indicate that they were of the type usually referred to as thyrotropic (progressive), but the authors' contention that progressive exophthalmos is a single entity which can occur in association with, or independently of, enlargement of the thyroid and thyrotoxicosis, would seem to be acceptable.

In evaluating the treatment of these cases it must be borne in mind that a single case report may be misleading in view of the tendency of the condition to be self-limiting.

In the treatment of thyrotoxicosis, thyroidectomy is justified if the urine shows little or no evidence of thyrotropic activity, the basal metabolic rate is high, the typical lid signs are present, the exophthalmos is minimal, and there are no congestive signs, lid edema, or chemosis, and no orbital resistance. It should be remembered that an apparent exophthalmos may be only a pseudoexophthalmos from lid retraction. In this connection it should be re-emphasized that the exophthalmos of thyrotoxicosis, which occurs in some 50 percent of cases only, is rarely over 3.0 mm., is usually not more than 1.5 mm., and is never progressive.

One authority states that in patients with toxic goiter presenting definite ocular protrusion, as opposed to mere lid retraction, it is wise to avoid thyroidectomy altogether. In these cases the use of iodine, the thiourea derivatives, and radioactive iodine may be of great value. In cases of marked lid retraction it may be necessary to do a lateral tarsorrhaphy to protect the cornea. This procedure may also be indicated for cosmetic improvement on psychologic grounds.

Thyroid therapy has been advocated for the treatment of progressive exophthalmos but most authorities regard it as ineffective and as definitely contraindicated in those cases in which a simultaneous excess of thyroxin and thyrotropin obtain. Thyroidectomy frequently aggravates progressive exophthalmos and is to be avoided. Other therapeutic measures, including stilbestrol, testosterone, cortisone, and ACTH, have been tried with no apparent success.

Radiation of the pituitary is at present receiving considerable attention. Some of the reports are encouraging but it has been stated that if the exophthalmos has existed for 18 months, X-ray therapy will probably fail. Evaluation of this therapeutic method must at all events wait on further experience with it.

If the exophthalmos has progressed until there is danger of corneal ulceration, or if there is loss of field or of vision due to edema or to stretching of the optic nerve, surgery is indicated. While there are those who advocate lateral tarsorrhaphy, the majority agree that the orbital decompression of Naffziger, while no panacea, is the procedure of choice.

Although there still exists a great deal of confusion regarding endocrine exophthalmos, the very extensive researches of recent years have illuminated the problem and given us an appreciably clearer understanding of it. One of the most important things we have learned is that exhaustive study of the case in hand may be decisive in indicating the therapeutic course that will preserve the patient's vision.

*384 Post Street (8).*

## References


1. Williams, R. H.: Textbook of Endocrinology. Philadelphia, Saunders, 1950.
2. Lerman, J., and Salter, W. T.: The relief of myxedema with proteins of extrathyroidal origin. Edocrinology, **25**:712-720, 1939.
3. Reineke, E. P., and Turner, C. W.: Thyroidal action of synthetic thyroprotein. J. Clin. Endocrinology, **3**:1-6, 1943.
4. v. Basedow, C.: Exophthalmos durch Hypertrophie des Zellgewebes in der Augenhöhle. Woch. f. d. ges. Heilk, Berlin, **14**:197-203, 1840.
5. Mulvany, J. H.: The exophthalmos of hyperthyroidism: Part I. Am. J. Ophth., **27**:589-611 (June) 1944.
6. Graves, R. J.: Newly observed affection of the thyroid gland in females. London M. and Surg. J., **7**:516-520, 1835.
7. Zondek, H.: In Kurzed Handbuch der ophthalmologie. Berlin, Julius Springer, 1932, v. 7, p. 375.
8. Naffziger, H. C.: Pathologic changes in the orbit in progressive exophthalmos, with special reference to alterations in the extra-ocular muscles and the optic disks. Arch. Ophth., **9**:1-12 (Jan.) 1933.
9. Tilley, J. H.: Exophthalmos: The mechanism of its production in exophthalmic goiter. Ann. Surg., **84**:647-650 (Nov.) 1926.
10. Moore, R. F.: Note on exophthalmos and limitation of eye movement in Graves' disease. Lancet, **2**:701, 1920.
11. Burch, F. E.: The exophthalmos of Graves' disease. Minnesota Med., **12**:668-675, 1929.
12. Moore, R. F.: Medical Ophthalmology. Philadelphia, Blakiston, 1922, p. 177.
13. Mulvany, J. H.: The exophthalmos of hyperthyroidism. Part I. Am. J. Ophth., **27**:589-611 (June) 1944.
14. ———: The exophthalmos of hyperthyroidism: Part III. Am. J. Ophth., **27**:820-832 (Aug.) 1944.
15. Sellers, E. A., and Ferguson, J. K. W.: Exophthalmos in rats after prolonged administration of propylthiouracil. Endocrinology, **45**:345-346 (Sept.) 1949.
16. Loeb, L., and Friedman, H.: Exophthalmos produced by infections of acid extract of anterior pituitary gland of cattle. Proc. Soc. Exper. Biol. & Med., **29**:648-650, 1932.
17. Marine, D., and Rosen, S. H.: Exophthalmos in thyroidectomized guinea pigs by thyrotropic substance on anterior pituitary and the mechanism involved. Proc. Soc. Exper. Biol. & Med., **30**:901-903, 1933.
18. Smelser, G. K.: Treatment of experimentally produced exophthalmos with thyroxin and other iodine compounds. Am. J. Ophth., **21**:1208-1218 (Nov.) 1938.
19. ———: Water and fat content of orbital tissues of guinea pigs with experimental exophthalmos produced by extracts of anterior pituitary gland. Am. J. Physiol., **140**:308-315 (Dec.) 1943.
20. ———: Study of retrobulbar tissues in experimental exophthalmos in guinea pigs with reference to primary and secondary modifications. Am. J. Anat., **72**:149-169 (Mar.) 1943.
21. ———: Reaction of orbital tissues in experimental exophthalmos following removal of Harder's gland. Anat. Rec., **85**:245-249 (Mar.) 1943.
22. ———: Oxygen consumption of eye muscles of thyroidectomized and thyroxin-injected guinea pigs. Am. J. Physiol., **142**:396-401 (Oct.) 1944.
23. ———: Changes induced in harderian gland of guinea pig by injection of hypophyseal extracts. Anat. Rec., **86**:41-57 (May) 1943.
24. ———: Differential concentration of hormones in central and peripheral zones of the bovine anterior pituitary gland. Endocrinology, **34**:39-43 (Jan.) 1944.
25. ———: Assay of thyrotropic hormone on day-old chicks. Proc. Soc. Exper. Biol. & Med., **37**:388-390 (Nov.) 1937.
26. Marine, D.: Studies on pathological physiology of exophthalmos of Graves' disease. Ann. Int. Med., **12**:443-453, 1938.
27. Brain, W. R.: Exophthalmos in Graves' disease despite sympathetic paralysis. Lancet, **2**:217, 1939.
28. Smelzer, G. K., and Ozanics, V.: Relation of steroid hormones to the development of experimental exophthalmos. Am. J. Ophth., **14**:87-94 (May) 1951.
29. ———: The reaction of various types of fat transplanted into the orbits of guinea pigs prior to the development of exophthalmos. Am. J. Ophth., **32**:1557-1563 (Nov.) 1949.
30. Jefferies, W. McK.: Studies of the relationship of the thyrotropic exophthalmic and fat mobilizing principles of the pituitary extract. J. Clin. Endocrinol., **9**:913-926 (Oct.) 1949.
31. ———: Studies of relationship of the thyrotropic: exophthalmic and fat mobilizing principles of the pituitary extract. The effect of iodination of pituitary extracts. Jour. Clin. Endocrinol., **9**:927-936 (Oct.) 1949.
32. Ludwig, A. W., Boas, N. F., and Soffen, L. J.: Role of Mucopolysaccharides in pathogenesis of experimental exophthalmos. Proc. Soc. Exper. Biol. & Med., **73**:137-140, 1950.
33. Asboe-Hansen, G., and Iversen, K.: Influence of thyrotropic hormone on connective tissue. Pathogenic significance of mucopolysaccharides in experimental exophthalmos. Acta Endocrinol., **8**:90-96, 1951.
34. Jefferies, W. McK.: Studies of the relationship of the thyrotropic, exophthalmic and fat mobilizing

principles of the pituitary extract: The effects of ACTH and Doca. J. Clin. Endocrinol., **9**:937-940 (Oct.) 1949.

35. Dayton, G. O., Jr.: The effect of ACTH on experimental exophthalmos. Am. J. Ophth., **35**:101-106 (May) 1952.

36. Aterman, K.: Cortisone and experimental exophthalmos. Lancet, **2**:1143-1144 (June) 1952.

37. Means, J. H., Hertz, S., and Williams, R. H.: Graves' disease with dissociation of thyrotoxicosis and ophthalmopathy. Tr. A. Am. Physicians, **56**:67-74, 1941.

38. Mann, I.: Exophthalmic ophthalmoplegia. Am. J. Ophth. **29**:654-673 (June) 1946.

39. Martens, T. G.: Exophthalmos of endocrine origin. Am. J. Med. Sc., **213**:241-245 (Feb.) 1947.

40. Braley, A. E.: The relationship between ocular disorders and thyroid dysfunction. J. Iowa State M. Soc., **38**:435-437 (Oct.) 1948.

41. Soffer, L. J.: Diseases of the Endocrine Glands. Philadelphia, Lea, 1951, p. 817.

42. Discussion on management of endocrine exophthalmos (Symposium) Proc. Royal Soc. Med., **45**:237-244 (Apr.) 1952.

43. Moore, R. F.: Medical Ophthalmology. Philadelphia, Blakiston, 1922, p. 177.

44. Woods, A. C.: Ocular changes of primary diffuse toxic goiter. Medicine **25**:113-153, 1946.

45. Dobyns, B. M.: Present concepts of the pathologic physiology of exophthalmos. J. Clin. Endocrinol., **10**:1202-1230 (Oct.) 1950.

46. Poppen, J. L.: The surgical treatment of progressive exophthalmos. J. Clin. Endocrinol., **10**:1231-1236 (Oct.) 1950.

47. Miles, E. S., and Forsey, R. R.: Localized myxedema associated with exophthalmos. Tr. A. Am. Physicians, **62**:225-232, 1949.

48. Grynkewich, S., Laughlin, R., Herron, F., and Carmel, W. J., Jr.: Localized myxedema in relation to thyrotoxicosis and exophthalmos. Am. J. Med. Sc., **222**:142-153, 1951.

49. Scheie, H., and Hedges, T. R.: Thyrotropic exophthalmos. New Orleans M. & Surg. J., **104**:481-485 (June) 1952.

50. Vail, D.: Exophthalmos. Postgraduate Med., **5**:439-450 (June) 1949.

51. Beierwalters, W. H.: Irradiation of the pituitary in the treatment of malignant exophthalmos. J. Clin. Endocrinol., **11**:512-530 (May) 1951.

52. Givner, I., Bruger, M., and Lowenstein, O.: Arch. Ophth., **37**: 211-219 (Feb.) 1947.

53. Braley, A. E.: Personal communication.

54. Brown, R. B., and Schwartz, G. A.: Optic neuritis in hyperthyroidism; report of a case with review of the literature. Arch. Inter. Med., **60**:668-679, 1937.

55. Walsh, F. B.: Clinical Neuro-ophthalmology. Baltimore, Williams & Wilkins, 1947, p. 817.

56. Mann, I.: Review: the orbital endocrine problem. Ophth. Lit., **1**:343-351 (Mar.) 1948.

57. Fuchs, E.: Textbook of Ophthalmology, (Translated by A. Duane.) Philadelphia, Lippincott, 1919, ed. 6, p. 806.

58. Frisk, A. R., and Josefsson, E.: Thiouracil derivatives and pregnancy. Acta Med. Scand., (Suppl.) **196**:85-91, 1947.

59. Iglesias, F. G.: Discussion of case. Am. J. Ophth., **34**:1043-1044 (July) 1951.

60. Medine, M. M. Malignant exophthalmos. Am. J. Ophth., **34**:1587-1590, (Nov.) 1951.

61. Dunnington, J. H., and Berke, R. N.: Exophthalmos due to chronic orbital myositis. Arch. Ophth., **30**:446-466 (Oct.) 1943.

62. Drescher, E. P., and Benedict, W. L.: Asymmetric exophthalmos. Arch. Ophth., **44**:109-128 (July) 1950.

63. Selinger, E.: Ocular signs and findings in exophthalmic goiter. Illinois M. J., **65**:319-324 (Apr.) 1934.

64. Rundle, F. F.: Some observations on exophthalmos. Western J. Surg., **55**:578-583 (Nov.) 1947.

65. Bedrossian, E. H.: Unilateral thyrotropic exophthalmos. Am. J. Ophth., **34**:727-732 (May) 1951.

66. Benedict, W. L.: Diagnosis of orbital tumors. J.A.M.A., **126**:880-884 (Dec.) 1944.

67. Saltzman, S. L., and Mellicker, M. C.: Unilateral exophthalmos as a forerunner of thyrotoxicosis. Am. J. Ophth., **34**:372-374 (Mar.) 1951.

68. Fischer, C.: Discussion of Parker, W. R.: Double luxation of the eyeballs in a case of exophthalmic goiter. Tr. Am. Ophth. Soc., **19**:134, 1921.

69. Gleason, J. E.: Idiopathic myositis involving the extraocular muscles. Ophth. Rec., **12**:471-475, 1903.

70. Falconer, M. A., and Alexander, W. S.: Experience with malignant exophthalmos. Relationship of the condition to thyrotoxicosis and to the pituitary thyrotropic hormone. Brit. J. Ophth., **35**:253-283 (May) 1951.

71. Plummer, H. S.: Results of administering iodine to patients having exophthalmic goiter. J.A.M.A., **80**:1955, 1923.

72. Astwood, E. B.: Treatment of hyperthyroidism with thiourea and thiouracil. J.A.M.A., **122**:78-81, 1943.

73. Jones, A.: Orbital X-ray therapy of progressive exophthalmos. Brit. J. Radiol., **24**:637-646 (Dec.) 1951.

74. Williams, R. H.: Textbook of Endocrinology. Philadelphia, Saunders, 1950, p. 145.
75. Cattell, R. B.: Surgical treatment of hyperthyroidism. J. Clin. Endocrinol., **9**:999-1006, 1949.
76. Dobyns, B. M.: Influence of thyroidectomy on prominence of eye in guinea pig and in man. Surg. Gynec. & Obst., **80**:526-533, 1945.
77. Beierwalters, W. H.: Exophthalmos and thiouracil therapy. Arch. Int. Med., **81**:364-368, 1948.
78. Soley, M. H.: Exophthalmos in patients with various types of goiter. Arch. Int. Med., **70**:206-220, 1942.
79. Soley, M. H., Miller, E. R., and Foreman, N.: Graves' Disease. J. Clin. Endocrinol, **9**:29-35, 1949.
80. Detroit, C.: Malignant exophthalmos during menopause. Acta Clin. belgica, **2**:240-244 (May-June) 1947.
81. Craig, J. D.: Malignant exophthalmos (two cases of successful treatment). J.A.M.A., **136**:946 (Apr.) 1948.
82. Thomas, H. M., and Woods, A. C.: Bull. Johns Hopkins Hosp., **59**:99-102, 1936.
83. Thomas, H. M., Jr.: Exophthalmos in the light of current anti-thyroid therapy. Am. J. Med., **11**: 581-587, 1951.
84. Soffer, L. J.: Diseases of the Endocrine Glands. Philadelphia, Lea, 1951, p. 821.
85. Hermann, K.: Pituitary exophthalmos: An assessment of the methods of treatment. Brit. J. Ophth., **36**:1-19 (Jan.) 1952.
86. Zonde, H., and Ticho, A.: Myasthenia gravis and malignant exophthalmos. Lancet, **2**:1018-1020 (Dec.) 1951.
87. Guyton, J. S.: Exophthalmos associated with endocrine dysfunction. Texas State J. Med., **43**:280-282 (Aug.) 1947.
88. Naffziger, H., Personal communication.
89. Laval, J., and Schneider, J.: Modified Snellen suture for prolapsed chemosed conjunctiva. Arch. Ophth., **38**:375-377 (Sept.) 1947.
90. Mann, I.: Endocrine disorders of the eye. Tr. Ophth. Soc. Australia, **11**:16-27, 1951.

---

# A NEW TREPHINING OPERATION

FREDERICK H. VERHOEFF, M.D., AND PAUL A. CHANDLER, M.D.
*Boston, Massachusetts*

## INTRODUCTION

Cutting a hole in the sclera is not the only important part of a trephining operation. Equally important is that part of the technique upon which largely depends persistence of the hole and continuance of adequate subconjunctival drainage.

The senior writer,* in 1936, described a technique which he still believes to be the simplest and best for attaining these ends. However, the technique involves the possible danger of the corneal suture pulling out, especially if it has not been well inserted. In the experience of the senior writer, this danger has been negligible but, about one year ago, to avoid it entirely, he devised a different technique. Although not as simple as the other, it is simpler and far less traumatizing than the technique described by Elliot and still generally employed.

The new features of the operation depend upon utilization of the elasticity of the conjunctiva to minimize the necessary dissection and to gain the best possible coverage for the trephining hole. The bulbar conjunctiva is grasped with forceps at a point six to eight mm. above the corneal limbus and pulled down until the conjunctiva overlaps the cornea four or five mm. Then a small opening, just large enough to admit the trephine blade, is made in the conjunctiva and Tenon's capsule just above the limbus. Through this opening the tissue is undermined at the scleral level down to the limbus, and the cornea is "split," if this further procedure is deemed advantageous. The sclera is then trephined, an iridectomy done, the con-

* Verhoeff, F. H.: A new conjunctival flap for trephining operations. Am. J. Ophth., **19**:46, 1936.

junctiva is allowed to retract and the hole in it is tightly closed by suturing.

### Present technique

Before the senior writer had actually performed this operation he described it to the junior writer who was so impressed by its obvious advantages that he began to employ it at his earliest opportunity. Following are some of the details in technique arrived at by his experience with the operation:

Anesthesia by retrobulbar injection is employed as in a cataract operation. This lowers the intraocular pressure. To restore the pressure to normal and, at the same time, deepen the anterior chamber, the latter is injected with normal salt solution, after first evacuating the aqueous through a beveled corneal incision made with a small knife.

To pull the conjunctiva down it should be grasped as superficially as possible. The best point at which to grasp it is determined by trial in each individual case and depends on the looseness and elasticity of the tissues. If a superior rectus bridle suture has been used, it should be completely loose at this stage so as to allow free mobility of conjunctiva and Tenon's capsule.

After the conjunctiva has been pulled down over the cornea, the forceps is transferred to the hand of an assistant who maintains the grasp. Then the operator grasps the conjunctiva farther back with another forceps and, with scissors, snips a small hole in the conjunctiva and underlying Tenon's capsule. The sclera is thus exposed directly above the limbus.

"Splitting" of the cornea may then be done if desired. To facilitate the splitting and to find the correct line of cleavage the assistant pulls the conjunctiva up at right angles to the cornea.

When the trephine opening is being made, the conjunctiva is again stretched down over the cornea. The sclera is trephined with a 1.5-mm. trephine and then a button-hole iridectomy is done. The conjunctival hole is closed with a catgut mattress suture.

Atropine solution is instilled, antibiotic ointment applied, and the eye bandaged. The bandage is removed the next day and the patient is discharged from the hospital, unless the anterior chamber is found abolished or the patient lives too far away.

### Results

Thus far the junior writer has performed the operation in five cases of chronic open-angle glaucoma. Bleeding during the operation was minimal. There appeared to be less postoperative reaction than after the conventional method. The operation required only five to 10 minutes.

In three eyes the anterior chamber was well formed during the postoperative period. In one eye it was flat at the first dressing but reformed in 48 hours. In one eye the anterior chamber remained flat for five days and was then caused to reform by release of the subchoroidal fluid and air injection into the chamber. This delay in reformation of the anterior chamber was not due to leakage through the conjunctival wound but, evidently, to subconjunctival drainage temporarily too abundant for this particular eye.

### Comment

These cases are, of course, too few and too recent to afford, in themselves, conclusive evidence as to the value of the operation. Nevertheless, since they reveal no features of the operation that are objectionable, and show that it can easily be carried out as planned, they make, in view of the obvious advantages of the operation, its early publication desirable.

Judging by the amount of traumatism to the tissue over and around the trephining hole, it is more than reasonable to expect that, in a large series of cases, the drainage would be better, the "blebs" less often vesicular, and hole closure less frequent, after the new than after the conventional operation.

*395 Commonwealth Avenue (15).*

# ARGYLL ROBERTSON PUPIL*

## FOLLOWING HERPES ZOSTER OPHTHALMICUS: WITH REMARKS ON THE EFFERENT PUPILLARY PATHWAYS

HOWARD A. NAQUIN, M.D.
*Baltimore, Maryland*

Pupillary constriction to light and accommodation-convergence (near reaction) have probably been recognized since antiquity. Credit is usually given to Father Paul of Venice[1] in the early 17th century for discovering the light reflex. Pupillary constriction to accommodation-convergence was very well described by Porterfield[1] in the 18th century.

In 1869[2] Douglas Argyll Robertson reported a series of cases with loss of the reflex to light but with preservation of constriction to accommodation-convergence. Since 1869, the observation has been repeatedly confirmed and is widely known as the Argyll Robertson pupil. An enormous literature has accumulated on the subject and there has been a great deal of controversy on the diagnostic significance of the abnormality and the anatomic site of the responsible lesion.

The loss of pupillary constriction to light with preservation of pupillary constriction to accommodation has been observed in a number of disease processes, and in a number of clinical variations. It has been reported in syphilis,[3] diabetes,[4] disseminated sclerosis,[5] third-nerve paralysis,[6] ocular trauma,[7] herpes zoster ophthalmicus,[8] and a number of other diseases.

It has been reported with small pupils, medium-sized pupils, and large pupils. It has been reported in unilateral and in bilateral form. There have been noted all degrees of loss of the light reflex. In some cases the constriction to light is totally absent while in others it may be only partially impaired. Likewise, the accommodation reflex may be present in various degrees.

A great deal of confusion has been brought on by the use of Argyll Robertson's name in describing abnormal pupils. A few physicians restrict the term Argyll Robertson pupil to a pupil having the characteristics described by Argyll Robertson. These include, in an eye with vision, the loss of pupillary constriction to light, with preservation of pupillary constriction to accommodation, miosis, and incomplete pupillary dilatation to atropine.

Others use the term to describe pupils with loss of constriction to light and preservation of constriction to accommodation, irrespective of pupillary size. Still others use the term to describe pupils where the reflex to light is more impaired than is the constriction to accommodation—a relative phenomenon.

Variation in light intensity used to elicit the light reflex and variation in the magnification used to observe pupillary constriction have introduced further confusion.

For some peculiar reason, nearly all authors have considered the afferent pathways carrying impulses from the retina to the third-nerve nucleus as the anatomic site of the responsible lesion and have given scant attention to the efferent pupillomotor pathways from the third-nerve nucleus to the iris as a possible site.

At the present time, most contemporary authorities[9] place the responsible lesion in the afferent pathways. On a theoretic basis, a lesion in the afferent pathways is perfectly plausible and, without doubt, there are diseases of the central nervous system which do interfere with the afferent pupillomotor pathways, producing pupillary light rigidity.

* From the Wilmer Ophthalmological Institute, The Johns Hopkins Hospital and University.

Likewise, on a theoretic basis, a lesion in the efferent pupillomotor pathways would offer a plausible explanation for the occurrence of a light-rigid pupil. In this case, one would have to assume two separate efferent pupilloconstrictor pathways—one for the light reflex and the other for the near reflex. This possibility has been suggested,[10] but has gained little recognition.

Kinier Wilson[11] has stated "the point of importance for the Argyll Robertson pupil is that distal to the oculomotor nucleus the pathways for iris contraction in response to light and in association with convergence-accommodation are identical." However, no clinical or experimental evidence to support this statement is offered.

It seems probable that the main reason so many authorities have insisted upon a lesion in the afferent pathways to explain a light-rigid pupil has been their failure even to consider the possibility of two efferent pupilloconstrictor pathways—one for the light reflex and another for the near reflex.

Even the advocates[12] of a lesion in the afferent pathways admit that there is little or no clinical or pathologic evidence to support their belief. On the other hand, there is much clinical evidence to support the belief that the efferent pathways are involved. Langworthy and Ortega[13] have summarized the evidence that the pupillary abnormalities seen in syphilis are peripheral rather than central. They too, however, failed even to consider the possibility of two separate efferent pupilloconstrictor pathways, and they were left therefore with no adequate explanation for the occurrence of pupillary light ridigity.

The occurrence of a light-rigid pupil following known involvement of the efferent pupillomotor pathways would constitute strong evidence in favor of two separate efferent pupilloconstrictor pathways.

Recently an opportunity has arisen to examine a patient, following herpes zoster ophthalmicus, who showed a light-rigid pupil conforming in every characteristic to the abnormality described by Argyll Robertson. This case is here reported in detail.

The various pupillary abnormalities which have been observed following herpes zoster ophthalmicus are discussed and evidence supporting the belief that all of these abnormalities are the result of involvement of the efferent pupillomotor pathways is presented. The similarity of the pupillary abnormalities following herpes zoster ophthalmicus and syphilis is pointed out.

Finally, a working hypothesis is presented which adequately explains all the various pupillary abnormalities, including the Argyll Robertson pupil, seen following herpes zoster and syphilis. Likewise, the hypothesis adequately explains the pupillary abnormalities occurring after injury and regeneration of the oculomotor nerve.

## Case report

G. M. (J.H.H. #291556), a 54-year-old Austrian-born machinist, was first seen in the Wilmer Clinic in May, 1943, complaining of pain and blisters over the right forehead and nose, of 13 days' duration.

*Examination* showed characteristic herpes zoster of the ophthalmic division of the right trigeminal nerve. Figure 1 is a photograph taken 14 days after onset of symptoms.

Corrected vision in the right eye was 20/200. The right eye showed marked conjunctival injection. The cornea showed superficial infiltrates, folds in Descemet's membrane, and numerous keratic deposits. The aqueous ray was faintly positive. The iris markings were "hazy" and the pupil was irregularly semidilated and fixed. (Atropine had been instilled prior to observation.)

The left eye had 20/20 corrected vision and was entirely normal.

The patient was hospitalized, and was treated with atropine locally, was given diphtheria antitoxen and was vaccinated with cowpox vaccine. The serologic test for syphilis was negative. The eye remained con-

gested for several months, but gradually improved.

Five months later, the eye was white. At that time the right pupil was described as somehat smaller than the left but no description of the pupillary reactions was recorded.

The patient was not seen here again until seven years later. The left eye was entirely normal. Corrected vision in both eyes was 20/15. There were healed scars over the right forehead and nose, with corresponding decreased sensation. The lids, lacrimal apparatus, extraocular movements, and conjunctiva were normal. There were some faint corneal scars on the right and corneal sensitivity was decreased.

The right pupil was irregular and flattened toward the 10-o'clock position and showed an atrophic radial scar of the iris. There was generalized iris atrophy with especially marked thinning of the stroma in the 10-o'clock meridian. The right pupil was smaller than the left, measured 1.5 by 1.75 mm. and did not react directly or consensually to light.

The left pupil was round, regular, and measured about 3.0 mm. in diameter. The left pupil reacted directly and consensually to light in an entirely normal manner.

Both pupils constricted briskly to convergence, both becoming approximately 1.25 mm. in size and both returned to their previous size rapidly when convergence was relaxed. There were no pupillary movements observed with vertical or horizontal changes in gaze. Accommodation was equal in both eyes, and measured 1.5 diopters.

Photographs of some of the pupillary reactions are shown in Figure 2. Figure 2-a shows the appearance of the two pupils in ordinary illumination. Miosis and irregularity of the right pupil are well shown. Figure 2-b shows the effect of light upon the right eye, and Figure 2-c the effect of light shone into the left eye. In both, the right pupil remained unchanged, while the left pupil constricted normally. Figure 2-d shows

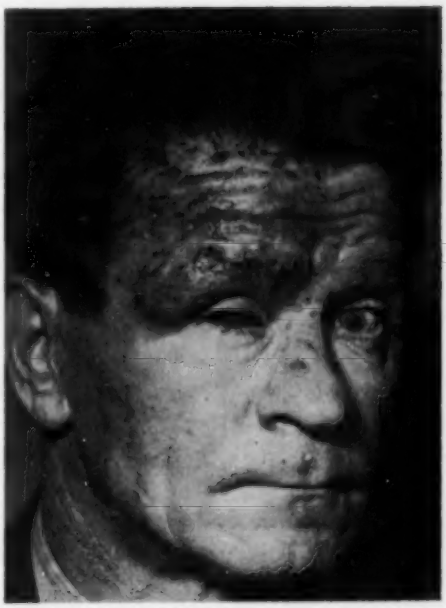


Fig. 1 (Naquin). Photograph taken 14 days after onset of symptoms.

the bilateral pupillary constriction upon convergence. Figure 2-e shows the incomplete dilatation of the right pupil two hours after instillation of one-percent atropine. The failure of the right pupil to constrict to light either directly or consensually was confirmed using the slitlamp.

The patient was seen at various times by members of the neurologic service and by members of the syphilis clinic, and no evidence of syphilis was found either by history, general physical examination, or neurologic examination. The blood serologic test for syphilis was negative on three occasions. Careful examination of the cerebrospinal fluid, including serologic tests, was entirely negative. Finally, the Treponema-immobilizing test[14] for syphilis was negative.

In summary, this case represents a case of true unilateral Argyll Robertson pupil, observed seven years after herpes zoster ophthalmicus, in a patient in whom syphilis can be completely excluded.

Fig. 2 (Naquin). *Flashbulb photographs of various pupillary reactions.* (a) Ordinary illumination. (b) Handlight illumination, right eye only. (C) Handlight illumination, left eye only. (d) Convergence reaction. (e) Two hours after instillation of one-percent atropine in each eye.

## Pupillary abnormalities following herpes zoster ophthalmicus

The occurrence of pupillary abnormalities following herpes zoster ophthalmicus has been known since the time of Jonathan Hutchinson.[15] The occurrence of Argyll Robertson pupils following herpes zoster ophthalmicus has also been known for many years, although the number of reported cases is comparatively few.

For purposes of discussion, it is convenient to divide the abnormal pupillary reactions seen after herpes zoster ophthalmicus into four arbitrary groups:

*The first group* is a group in which the pupillary abnormality consists primarily of paralysis of the sympathetic pupillodilator fibers.

*The second group* consists of pupillary abnormalities associated with third-nerve paralysis. In this group the parasympathetic pupilloconstrictor fibers are primarily affected.

*The third group* consists of pupillary abnormalities reported as Argyll Robertson pupils.

*The fourth group* consists of various combinations of sympathetic and parasympathetic involvement not included in the other three groups.

Each group in turn is here considered and the clinical evidence is summarized which supports the view that, for each group, the efferent pupillomotor pathways are affected.

### 1. Sympathetic paralysis

There are a number of cases reported of Horner's syndrome following herpes zoster ophthalmicus. Bing,[16] in his textbook of neurology, described and showed a photograph of a young girl who developed miosis, enophthalmos, and ptosis at the same time as she developed typical herpes zoster ophthalmicus.

Jacquet and Bariety[17] reported a similar case, but without miosis. They noted, moreover, that the pupil dilated poorly to atropine.

Potts[18] reported a case of Horner's syndrome without disturbance of facial sweating following ophthalmic zoster. The pupil did not dilate to cocaine, and Potts considered that the sympathetic pathways were affected peripherally in the region of the Gasserian ganglion, after the fibers that control sweating of the face had been given off. He thought the case represented one of the paratrigeminal syndrome, as described by Raeder[19] two years previously. Recently, Grignolo[20] has reported another case, and from the responses of the pupil to various

drugs, localized the lesion in the most peripheral sympathetic neuron, between the superior cervical ganglion and the iris itself.

These cases and others[21] in the literature are suggestive of peripheral involvement of the sympathetic pathways and cannot be explained adequately by involvement of the sympathetic pathways in the central nervous system.

### 2. Pupillary abnormalities associated with third-nerve paralysis

The second group includes pupillary abnormalities associated with third-nerve paralysis. Partial or complete oculomotor paralysis is known to be associated with herpes zoster ophthalmicus, and it is obvious that the resulting pupillary changes may be identical with third-nerve paralysis of any etiology.

There are many reports[22] of dilated, completely immobile pupils following oculomotor paralysis associated with zoster. These cases are easily explained on the basis of paralysis of the efferent pupilloconstrictor pathways in the oculomotor nerve.

Bonnet and Chauvire[23] reported a case of herpes zoster ophthalmicus associated with oculomotor paralysis. About five months after the acute attack and during the recovery from the oculomotor paralysis, they noted that the pupil was moderately dilated, was fixed to light, but reacted to convergence-accommodation.

It has been known for many years that following partial or complete paralysis of the oculomotor nerve, either by injury within the orbit or the cranium, the pupil may become fixed to light but still react normally to accommodation. There have been various attempts to explain this unusual fact.

Nathan and Turner,[10a] among others, have suggested that there must be two separate efferent pupillconstrictor pathways. Their report has not received the attention it deserves. Ford, Walsh, and King[41] have assumed one efferent pupilloconstrictor pathway, with synapse in the ciliary ganglion and suggested that due to the small number of these fibers, they failed to reachieve synapse during regeneration, while the fibers formerly destined for the extraocular muscles, through their relatively large number, achieved synapse in the ciliary ganglion and were able to cause iris constriction.

Bender[6a] also assumed one efferent pupilloconstrictor pathway and suggested that the stimulus of light sent a relatively weak impulse down that pathway and that the impulse was blocked at the site of injury, while the act of convergence sent a strong impulse down the same pathway enabling the impulse to get past the injury and reach the iris.

Another possibility is the assumption of two efferent pathways for pupillary constriction, one for the light reflex, with synapse in the ciliary ganglion and the other for the accommodation-convergence reaction, passing from the midbrain without synapse to the iris sphincter. With two entirely different types of nerve fibers, it would then not be unusual if they reacted differently to injury either by disease or trauma. The phenomenon of synkinetic pupillary constriction consisting of pupillary constriction upon lateral or vertical gaze observed experimentally and in humans with third-nerve regeneration and misdirection suggests that the iris sphincter is able to constrict to impulses from motor nerves with cell bodies in the midbrain.

### 3. Argyll Robertson pupil

Lapersonne and Cantonnet,[24] in 1909, reported a case of unilateral Argyll Robertson pupil associated with sympathetic paralysis on the same side. Their patient was a 30-year-old woman, who had noted unequal pupils following an acute illness at the age of 11 years, diagnosed as erysipelas. Although the authors do not mention the possibility of zoster, it seems likely from the report that this may have been the true etiology. One pupil was small, slightly irregular, and did not react to light either directly

or consensually. The opposite pupil was entirely normal. Both pupils reacted to accommodation.

Apparently the first recognized case of an Argyll Robertson pupil following herpes zoster was reported by Verhoeff[34a] in 1919. In discussing a paper on ocular complications of herpes zoster before the American Ophthalmological Society, he reported observing a "typical unilateral Argyll Robertson pupil" following ophthalmic zoster. The Wassermann reaction was negative, and there were no signs of syphilis. Verhoeff stated that this case strongly supported the view that the Argyll Robertson pupil was due to a lesion of the ciliary ganglion.

In 1945, after an exhaustive search of the literature, Edgerton[35] reported that among the approximately 2,250 cases of herpes zoster ophthalmicus he had found in the literature, there were reported[a] 23 cases of Argyll Robertson pupils. This is the only figure in the literature on the incidence of this abnormality.

A case of particular interest was reported by Redslob[36b] in 1923. Three months after herpes zoster ophthalmicus, the patient showed an irregular slightly dilated pupil which did not react to light, either directly or consensually. Upon convergence-accommodation only the nasal half of the iris constricted. The iris showed marked atrophy especially temporally. Redslob considered that the inflammatory process of herpes had reached the ciliary ganglion via the sensory branches of the trigeminal and had destroyed the motor elements of the ganglion. He also stated that this case proved that convergence had a more powerful action than light on the contraction of the pupil. He did not consider the possibility of two efferent pupilloconstrictor pathways.

Most of the authors reporting cases have noted that the abnormal pupillary reactions had their onset sometime after the acute phase of the zoster. In only one case, however, were the data sufficient to date the onset accurately.

François[8k] reported an extremely well-followed case of herpes zoster ophthalmicus in a 23-year-old man. Five days after the onset of the skin eruption, both pupils were equal and reacted entirely normally. Two days later, the iris became congested and the pupillary reaction became weaker. Atropine was started and continued for four months.

Three weeks after discontinuing atropine and five months after onset of zoster the pupil was moderately dilated, and did not react to either light or accommodation. Three weeks later, the pupil remained fixed to light, but constricted distinctly to accommodation. It measured four mm. in diameter. Seven months later, the pupillary reactions remained unchanged.

At no time did the patient show evidence of extraocular muscle paralysis. The opposite pupil was entirely normal at all times. This case is remarkable in showing that while both the light reflex and the near reflex were rendered nonfunctional by the herpetic process, the accommodation reflex was in some manner able to re-establish itself.

### 4. Other pupillary abnormalities

The fourth group of pupillary abnormalities takes in the remainder not included in the other three groups. Jonathan Hutchinson[15] noted cases where the pupil did not dilate to atropine following herpes zoster. He described one case in which, two months following herpes of the forehead, lids, and nose, one pupil was twice as large as the other and absolutely motionless both to direct and reflex stimulation. He noted that the pupil was oval shaped, and that the iris was not visibly inflamed and that there were no visible adhesions.

Since Hutchinson's time, there have been many similar cases reported[2a] in the literature. In most of them, the pupil has been dilated, either regularly or irregularly, and the reactions to light and near have been completely absent or partially absent.

Associated paralysis, either partial or complete, of the ciliary muscle has been reported. Associated atrophic iris changes and partial or complete failure of the pupil to dilate with atropine have likewise been reported. Most of the authors have postulated a herpetic lesion involving the ciliary ganglion. This group of pupillary abnormalities is adequately explained by postulating peripheral involvement of the pupillomotor pathways.

## Comment

All types of pupillary abnormalities reported following herpes zoster, including the Argyll Robertson pupil, have several things in common. They invariably follow herpes zoster of the ophthalmic division of the trigeminal and they are invariably unilateral and ipsilateral. The importance of these facts in localizing the anatomic site of the responsible lesion is apparent. It is obvious that were the abnormalities the result of involvement of the central pathways by the spread of the inflammatory process in spinal fluid or brain parenchyma, it would be fantastic to have that involvement always unilateral and ipsilateral and occurring only after herpes of the first branch of the trigeminal.

The available pathologic evidence indicates that the involvement of the pupillary pathways is in the efferent pathways. Since von Barensprung's original observation in 1861,[27] the majority[28] of pathologic examinations in herpes zoster ophthalmicus have shown the primary lesion to be in the Gasserian ganglion. The changes found consist of acute, round-cell inflammation with hemorrhage in the ganglion, destruction of ganglion cells and fibers, and inflammation of the sheaths of the ganglion. The nerve fibers connected with the ganglion cells show degenerative changes extending to their peripheral end-organs.

Meller[29] reported perineuritis of the ciliary nerves after herpes zoster ophthalmicus. There frequently[25] are pathologic changes in the eye, including the iris, that are visible grossly both during and after herpes zoster ophthalmicus.

The sensory afferent fibers going from the eye to the Gasserian ganglion are in intimate association with the efferent pupillomotor pathways, sympathetic and parasympathetic, and it would be quite likely that an inflammatory process involving one set of fibers could also affect the other. Likewise, one might expect various gradations and combinations of involvement of the sympathetic and parasympathetic pupillomotor fibers. We have already seen that such is actually the case.

The abnormal reactions of the pupil to various drugs following herpes zoster ophthalmicus are entirely explicable on the basis of peripheral involvement of the pupillary pathways, and are completely incompatible with a central lesion. The irregularity in shape of the pupil found after herpes zoster is indicative of an efferent lesion and is incompatible with a lesion in the afferent pupillary pathways.

The occurrence of constriction of one half of the iris only, as reported by Redslob, is completely incompatible with a lesion in the afferent pathway. While each item of evidence by itself might be challenged as being an incidental finding in association with a central lesion, taken together, they constitute overwhelming evidence against a central lesion.

If one will admit that the pupillomotor fibers are involved peripherally by herpes zoster, then my case and the cases I have briefly reviewed are positive proof that there must be at least two separate efferent pathways for pupillary constriction, one for the light reflex and the other for the accommodation-convergence reflex. Moreover, if there are two pathways, there must be some fundamental difference in the reaction of each pathway to disease processes in order to explain the extreme frequency of isolated pupillary light rigidity in relation to the extreme rarity of isolated convergence-accommodation rigidity. The case of François suggests that one aspect of that fundamental difference is the greater ability of the convergence-accommodation fibers to re-estab-

lish their function following injury.

The occurrence of unilateral pupillary light rigidity is sometimes used as an argument that the responsible lesion must be in the efferent pupillomotor pathways. This is not necessarily true. The only diagnostic significance of true unilateral pupillary light rigidity is that the responsible lesion must be either in the efferent pathways or in the afferent pathways posterior to the most posterior decussation of the afferent fibers. There is evidence of at least three sites of decussation in the afferent pathways—the optic chiasm, the posterior commissure, and in the midbrain ventral to the central gray matter enclosing the rostral end of the cerebral aqueduct.

The knowledge of these decussations plus the known occurrence of unilateral pupillary light rigidity has been the principal reason for selecting the region just lateral to the aqueduct of Sylvius as the anatomic site for the responsible lesion in syphilitic pupillary light rigidity. It should be noted that there is no pathologic evidence whatsoever to confirm this idea.

Likewise, it should be noted that it is necessary to postulate bilateral lesions in the afferent pupillary pathways to explain the occurrence of bilateral pupillary light rigidity, just as it is necessary to postulate bilateral lesions in the efferent pupillary pathways to explain bilateral pupillary light rigidity. No single unilateral lesion either in the afferent or efferent pathways will explain bilateral pupillary light rigidity.

The fact that pupillary abnormalities are usually bilateral in syphilis is therefore, in itself, not of any localizing value. It merely reflects the fact that the Treponema pallida usually affects both sides of its host. The fact that the pupillary light rigidity following herpes zoster is unilateral is likewise, in itself, not indicative of either afferent or efferent pupillomotor involvement. Rather, it reflects the peculiar localization of the herpetic process to one side of the symmetrical human organism.

The similarity between the pupillary abnormalities occurring after herpes zoster and those occurring in association with syphilis is remarkable. In both conditions are found irregular, dilated pupils, completely inactive or only partially active to both light and accommodation. In both are found large pupils which do not react to light, but react normally to accommodation. In both are found irregular pupils, with atrophic changes in the irises. In both are found typical Argyll Robertson pupils. Abnormal pupillary reactions to drugs, indicative of peripheral involvement of the autonomic nerves, are found in both. Both may show involvement of the sensory fibers of the trigeminal. In both there is a conspicuous absence of reports of cases showing loss of the accommodation reflex with preservation of the light reflex.

The single striking difference between the two is the invariable unilaterality of the pupillary abnormalities after herpes zoster, and the usual bilaterality after syphilis.

It seems extremely likely that the anatomic site of the lesion responsible for the pupillary abnormalities is the same in the two diseases. In view of the complete lack of any positive evidence in favor of a central site for the lesion causing syphilitic pupillary abnormalities, including the Argyll Robertson pupil, there should be no reluctance in discarding that concept.

## Conclusion

The evidence, from the case I have reported and from a review of the literature, is overwhelming that all the pupillary abnormalities that occur following herpes zoster ophthalmicus are the result of peripheral involvement of the sympathetic and parasympathetic fibers to the eye. The occurrence of loss of the pupillary reflex to light with preservation of the pupillary reflex to convergence-accommodation following herpes zoster is proof that there must exist at least two separate efferent pathways for pupillary constriction.

Not only is there evidence for two separate pathways, but there is evidence of a fundamental difference in the reaction of these two pathways to disease processes. The exact nature of this difference is not known, and must await further investigation, but the available evidence indicates that one aspect of this difference may be the greater ability of the convergence-accommodation pathway to re-establish itself following disease or injury.

While awaiting more precise neuroanatomic information as regards these pathways, one may conveniently explain all clinical observations by postulating that the constrictor fibers from the convergence-accommodation center in the midbrain pass without synapse to the iris sphincter, whereas the constrictor fibers for the light reflex synapse in the ciliary ganglion before reaching the iris sphincter.

*The Johns Hopkins Hospital (5).*

## References

1. Porterfield, W.: A Treatise on the Eye. Edinburgh, J. Balfour, 1759.

2. a. Robertson, D. Argyll: On an interesting series of eye symptoms in a case of spinal disease with remarks on the action of belladonna on the iris, etc. Edin. M. J., **14**:696-708, 1869.

b. ———: Four cases of spinal miosis with remarks on the action of light on the pupil. Edin. M. J., **15**:487-493, 1869.

3. a. Walsh, F. B.: Clinical Neuro-ophthalmology. Baltimore, Williams & Wilkins, 1947.

b. Adie, W. J.: Argyll Robertson pupils true and false. Brit. M. J., **2**:136-138, 1931.

4. a. Major, R. H.: Tabes diabetica. J.A.M.A., **83**:2004, 1924.

b. Wakefield, E. G.: Diabetic neuritis. Proc. Staff Meet. Mayo Clinic, **3**:256, 1928.

c. Waite, J. H. and Beetham, W. P.: The visual mechanism in diabetes mellitus: A comparative study of 2,002 diabetics and 457 nondiabetics for control. New Eng. J. M., **212**:367, 429, 1935.

d. Jordan, W. R.: Neuritic manifestations in diabetes mellitus. Arch. Int. Med., **57**:307, 1936.

e. Joslin, E. P., Root, H. F., White, P., and Marble, A.: Treatment of diabetes Mellitus. London, Kimpton, 1940.

f. Rundles, R. W.: Diabetic neuropathy: General review with report of 125 cases. Medicine, **24**:111-160, 1945.

g. Smith, M.: Diabetic neuropathy with Argyll Robertson pupils; report of 2 cases. Glasgow M. J., **30**:181-184, 1949.

5. a. Uhtoff, W.: The significance of eye symptoms in disseminated sclerosis of the brain and spinal cord. Ophthalmoscope, **3**:429, 1905.

b. Abramson, J. L., and Teitelbaum, M. H.: The Argyll Robertson phenomenon in multiple sclerosis. Am. J. Ophth., **16**:676, 1933.

6. a. Hutchinson, J.: Notes on the symptom significance of different states of the pupil. Brain, **1**:1, 1878.

b. Gowers: Diseases of the Nervous System. London, J & A Churchill, v. 2, p. 173, 1888.

c. Axenfeld, T.: Deutsche med. Wchnschr., **32**:663, 1906.

d. Abelsdorf: Neurol. Centralb., **25**:285, 1906.

e. Thompson, A. H.: Overaction of levator palpebrae on attempting to look down or in with a partially paralyzed inferior rectus. Tr. Ophth. Soc. U. Kingdom, **26**:207, 1906.

f. Rose, F.: A R sign in nonsyphilitic disease. Semaine Med., **32**:577, 1912.

g. Velter, E.: Signe d'Argyll-Robertson unilateral par traumatisine orbitaine. Arch. d'ophtal., **33**:120, 1913.

h. Bollack, J.: Signe d'Argyll Robertson unilateral par eclat d'obus intraorbitaire. Arch. d'ophtal., **36**: 106, 1918.

i. Lagrange, H., and Lagrange, A.: Argyll Robertson pupils. Ann. d'ocul., **172**:631, 1935.

j. Harris, W.: The fibers of the pupillary reflex and the A-R pupil. Arch. Neurol. & Psychiat., **34**: 1195, 1935.

k. Bender, M. B., and Alpert, S.: Abnormal ocular and pupillary movements following oculomotor paralysis. Arch. Ophth., **18**:411-414, 1937.

l. Ford, F. R., Walsh, F. B., and King, A. B.: Clinical observation on the pupillary phenomenon resulting from regeneration of the third nerve with especial reference to the A-R pupil. Bull. Johns Hopkins Hosp., **68**:309-318, 1941.

m. Nathan, P. W., and Turner, W. A.: Efferent pathway for pupillary contraction. Brain, **65**:343-351, 1942.

n. Cristini, G.: Anomalous associated palpebral and pupillary movements due to faulty regeneration of fibers of common oculomotor nerve. Rev. oto-neuro-oftal., **22**:323, 1947.

o. Schretzenmayr, V.: Associated movements of eyelids and pupils in oculomotor paralysis. Deutsche Ztschr. Nervenh., **158**:43, 1947.

p. Russell, W. R., and Wright, M. H.: Case report: Abnormal innervation of the sphincter pupillae and ciliary muscle following third nerve regeneration. J. Neurol. Neurosurg. & Psychiat., **11**:288, 1948.

q. Bender, M. B.: Synkinetic pupillary phenomenon and the A-R pupil. Arch. Neurol. & Psychiat., **53**: 418, 1945.

7. a. Magnani, C.: Clinical contributions to the study of the innervation of the iris. Arch. Ophth., **33**: 591, 1904.

b. Cosmettatos, G. F.: Unilateral loss of the light reflex with preservation of the Accommodation reflex of traumatic origin. Arch. d'ophtal., **25**:664, 1905.

c. Vincent: Societe de Neurologie. June, 1914. (Cited by Bollock, J.)

d. Magitot, A.: Sur quelques variations traumatique de la tension oculaire. Ann d'ocul., **154**:667, 1917.

e. Dufour, H.: Societe Neurologie. February, 1917. (Cited by Bollock, J.)

8. a. Verhoeff, F. H.: Discussion. Am. J. Ophth., **2**:761, 1919.

b. Redslob, E.: Pupillary abnormalities in ophthalmic zona. Rev. d'oto-neuro-ocul., **1**:509, 1923.

c. Guillain, G., Lagrange, H., and Perisson, J.: Signe d'Argyll-Robertson unilateral consecutif a un zona ophtalmique. Bull. Soc. d'ophtal. Paris, 1926, p. 227.

d. Renard, G.: Troubles pupillaries et zona. Bull. Soc. d'ophtal. Paris, **40**:555, 1928.

e. Cousin, G., and Perisson, J.: A new case of unilateral Argyll Robertson pupil following ophthalmic zona. Rev. Neurol., **52**:150, 1929.

f. Valiere-Vialeix, V.: Frequency of parenchymatous keratitis in ophthalmic zona: New case of unilateral Argyll Robertson sign following ophthalmic zona. Ann. d'ocul., **168**:341, 1931.

g. Wolff, E.: Discussion following Neame, H.: Affections of the eye due to virus. Tr. Ophth. Soc. U. Kingdom, **51**:24, 1931.

h. Satanowsky, P.: Frequency of parenchymatous keratitis, unilateral Argyll Robertson pupil, cyclitis, and glaucoma following ophthalmic zona. La Semana Medica, **52**:1919, 1931.

i. Hermann, P.: Signe d'Argyll-Robertson persistent trois ans apres un zona ophtalmique. Bull. Soc. d'ophtal. Paris, **226**, 1935.

j. Milian, G., and Chapireau: Ophthalmic zoster and the unilateral sign of Argyll Robertson. Bull et mem. Soc. med. d. hosp. Paris, **51**:1743, 1935.

k. François, J.: Signe d'Argyll Robertson et zona ophtalmique. J. Belge neurol. et Psychiat., **36**:17, 1936.

l. Ciotola, G.: Boll. d'ocul., **15**:1294, 1936.

m. Zutt, J.: Unilateral pupillary light paralysis after severe herpes zoster in the territory of the first and second divisions of the trigeminal. Monatsbl. f. Psychiat. u. Neurol., **93**:305, 1936.

n. Kissel, P., Cordier, J., and De Ren, G.: True unilateral Argyll Robertson pupil after ophthalmic zona. Rev. med. Nancy, **72**:173-177, 1947.

o. Luhan, J. A.: Postherpetic Argyll Robertson pupil. Arch. Neurol. and Psychiat., **59**:839, 1948.

p. Cordier and Pissavin: Sign of Argyll Robertson and ophthalmic zona. Ann. d'ocul. **181**:573, 1948.

9. a. Merritt, H. H., and Moore: Argyll Robertson pupil. Arch. Neurol. & Psychiat., **30**:357, 1933.

b. Wilson, S. A. K.: Neurology. Baltimore, William & Wilkins, 1940, v. 1, p. 483.

c. Duke-Elder, S.: Textbook of Ophthalmology, St. Louis, Mosby, 1942, p. 571.

d. Rea, R. L.: Neuro-ophthalmology. St. Louis, Mosby, 1941, p. 22.

e. Bing, R., and Haymaker, W.: Regional diagnosis in lesions of the brain and spinal cord. St. Louis, Mosby, 1940, p. 228.

f. Woods, A. C.: Syphilis of the eye. Am. J. of Syph., Gonor., & Ven. Dis., **27**:133-186, 1943.

g. Spiegel, E. A., and Sommer, I.: Neurology of the Eye, Ear, Nose, and Throat. New York, Grune and Stratton, 1944, pp. 444-448.

h. Kestenbaum, A.: Clinical Methods of Neuro-ophthalmologic Examination. New York, Grune and Stratton, 1946, p. 292.

i. Merritt, H. H., Mettler, F. A., and Putnam, T. S.: Fundamentals of Clinical Neurology. Philadelphia, Blakiston, 1947, p. 20.

j. Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice. Baltimore, Williams & Wilkins, 1950.

k. Berens, C.: The Eye and Its Diseases. Philadelphia, Saunders, 1949, ed. 2.

l. Town, A. E.: Ophthalmology. Philadelphia, Lea, Febiger, 1951, p. 402.

m. Adler, F. H.: Gifford's Textbook of Ophthalmology. Philadelphia, Saunders, 1947, ed. 4, p. 276.

n. Stallard, H. B.: Modern Practice in Ophthalmology. London, Butterworth, 1949, p. 89.

o. Sorsby, A.: Systemic Ophthalmology. St. Louis, Mosby, 1951, p. 237.

p. Cogan, D. G.: Neurology of the Ocular Muscles. Springfield, Ill., Thomas, 1948.

10. a. Ohm, J.: A case of unilateral pupillary light rigidity with preservation of convergence reaction following oculomotor paralysis in the orbit. Centralb. f. Augenh., **31**:193, 1907.

b. Foerster, O., Gagel, O., and Mahoney, W.: The anatomy, physiology, and pathology of pupillary innervation. Verhandl. deutsch. int. Med. Gesellsch., **48**:386, 1936.

c. Nathan, P. W., and Turner, J. W. A.: Efferent pathways for pupillary contraction. Brain, **65**:343-351, 1942.

11. Wilson, S. A. K.: The Argyll Robertson pupil. J. Neurol. & Psychopathol., **2**:1, 1921.

12. Merritt, H. H., Adams, R. D., and Solomon, H. C.: Neurosyphilis. New York, Oxford University Press, 1946, p. 263.

13. Langworthy, O. R., and Ortega, L.: The Iris. Medicine, **22**:287-361, 1943.

14. a. Nelson, R. A., Jr., and Mayer, M. M.: Immobilization of treponema pallidum in vitro by antibody produced in syphilitic infection. J. Exper. Med., **89**:369, 1949.

b. Magnuson, H. J., and Thompson, F. A., Jr.: Treponemal immobilization test of normal and syphilitic serums. J. Ven. Dis. Inform., **30**:309, 1949.

15. a. Hutchinson, J.: A clinical report on herpes zoster frontalis seu ophthalmicus (shingles affecting the forehead and nose) Roy. London Ophth. Hosp. Rep. **5**:191-215, 1866.

b. ———: A second report on herpes zoster frontalis. Roy. London Ophth. Hosp. Rep. **6**:181-189, 1869.

16. a. Bing, R.: Textbook of Nervous Diseases. New York, Rebman, 1915.

b. Bing, R., and Haymaker, W.: Textbook of Nervous Diseases. St. Louis, C. V. Mosby, 1939, ed. 5.

17. Jacquet, P., and Bariety, M.: Bull. et Mem. Soc. med. d. Hop. Paris, **50**:1561, 1926.

18. Potts, C. S.: Paralysis of the oculopupillary sympathetic fibers and the ophthalmic division of the trigeminal nerve, preceded by ophthalmic herpes zoster. Arch. Neurol. & Psychiat., **16**:71, 1926.

19. Raeder, J. G.: "Paratrigeminal" paralysis of the oculo-pupillary sympathetic. Brain, **47**:149, 1924.

20. Grignolo, A.: A Case of Claude-Bernard-Horner's syndrome following ophthalmic zoster. Rev. oto-neuro-oftal., **23**:387, 1948.

21. Aubaret and Morenon: Rev. Neurol., **48**:201, 1927.

22. a. Rutter: Note on ocular paralysis following herpes facialis. Lancet, **1**:298, 1899.

b. Sulzer, G. A.: Herpes zoster ophthalmicus. Ann. Ophth., **16**:120, 1907.

c. Fage: Rare complications of zona ophtalmique. Rev. d'ophth., **30**:209,1909.

d. Hunt, J. R.: The paralytic complications of herpes zoster of the cephalic extremity. J.A.M.A., **53**: 1456, 1909.

e. Chance, B.: Herpes zoster ophthalmicus. Ann. Ophth., **24**:693, 1915.

f. St. Martin: Oculomotor complications of zona ophthalmic; Trophic disorders of the iris; zona and varicella. Bull. Soc. d'ophtal. Paris, **41**:39, 1929.

g. Pincus, M. H.: Herpes zoster ophthalmicus. Am. J. Ophth., **32**:130, 1949.

23. Bonnet, P. and Chauvire, E.: Ophthalmic zona: False Argyll Robertson pupil during the decline of a paralysis of the oculomotor nerve. J. d'ophth., **2**:265, 1943.

24. de Lapersonne, F., and Cantonnet, A.: Signe d'Argyll Robertson unilateral avec coesistence, du meme cote, d'un syndrome oculo-sympathique incomplet. Rev. Neurol., **18**:1515, 1909.

25. Edgerton, A. E.: Herpes zoster ophthalmicus. Arch. Ophth., **34**:40, 1945.

26. a. Jessop, W. H.: On the affections of the eye in herpes facialis, with cases. Tr. Ophth. Soc. U. Kingdom, **6**:390, 1886.

b. Dufour: Mydriasis in the course of zona ophthalmica. Ann. d'ocul., **136**:139, 1906.

c. Elliot, R. H.: Some observations on herpes zoster ophthalmicus. Tr. Ophth. Soc. U. Kingdom, **38**: 351, 1918.

d. Aurand: Mydriasis following zona ophthalmica. Lyon Med., **129**:458, 1920.

e. Baranov, M.: Herpes zoster ophthalmicus with internal ophthalmoplegia. Tr. Ophth. Soc. U. Kingdom, **42**:167, 1922.

f. Bar, A.: Sequellae of ophthalmic Zona. Klin. Monatsbl. f. Augenh., **75**:393, 1925.

g. Valiere-Vialeix, Prosper-Veil, and Isnel, R.: On complications usually considered rare in ophthalmic zona. Ann. d'ocul., **162**:569, 1925.

h. Genet, L.: Mydriasis and fixed pupil following ophthalmic zoster. Lyon Med., **136**:51, 1925.

i. Ligertwood, L. M.: Mydriasis and cycloplegia as a result of herpes ophthalmicus. Edin. M. J., **34** 423, 1927.

j. Federici: Complications rares du zona ophtalmique. Boll. d'ocul., 1929.

k. Braun: Oculomotor complications of ophthalmic zoster. Thèse, Paris, 1930.

l. Rollet, J., Bujadoux, and Blanc.: Zona ophtalmique avec complications oculaires. Soc. d'ophtal. de Lyon, March, 1931, p. 21.

m. Genet, L.: Irregular, fixed pupils and abnormal pigmentation of the iris in ophthalmic zona. Bull Soc. d'ophtal. Paris, 1934, p. 234.

n. François, J.: Zona ophtalmique et paralysis oculaires. J. belge de neurol. et psychiat., **36**:8, 1936.

27. Von Barensprung: Ann. d. char. Krankenh., **9**:40, 1861.

28. a. Head, H., and Campbell, A. W.: Pathology of herpes Zoster and its bearing on sensory localization. Brain, **23**:353, 1900.

b. Wyss, O. Arch. f. Heilk., **16**:261, 1871.

c. Paton, L.: The trigeminal and its ocular lesions, Brit. J. Ophth., **10**:305, 1926.

29. Meller, J.: Ueber die Perineuritis und periarteritis ciliaris bei Herpes Zoster Ophthalmicus. Ztschr. f. Augenh., **50**:2, 1923.

# THE SIGNIFICANCE OF THE UNILATERAL ARGYLL ROBERTSON PUPIL*

## PART I. A REPORT OF 13 CASES

JULIA T. APTER, M.D.
*Chicago, Illinois*

The unilateral Argyll Robertson syndrome is regarded as a rarity. Merritt and Moore's review[41] of the hospital records of 749 tabetic patients is quoted widely to substantiate this view. They found that eight (1.1 percent) of these patients had unilateral Argyll Robertson pupils, while 226 (30 percent) had the bilateral Argyll Robertson syndrome.

In addition, Lutz,[37] Casper,[7,8] Jessop,[29] Frenkel,[17] Menninger,[40] Ingvar,[26] and Morelli,[44] each reported single cases of unilateral, miotic, light-rigid pupils in patients with neurosyphilis. These separate reports imply that the condition is rare.

On the other hand, Clark,[10] Jaeger,[28] Shapira and Crage[53] have reported that unilateral, miotic, light-rigid pupils occur more frequently than in 1.1 percent of patients with neurosyphilis. Moreover, Merritt and Moore point out that the criteria for the diagnosis of Argyll Robertson pupil were seldom defined in the charts from which they collected their statistics.

The present study with the slitlamp biomicroscope directs attention to a relatively high incidence of unilateral Argyll Robertson pupils. Since a high incidence of unilateral, miotic, light-rigid pupils in neurosyphilis makes untenable the present theories of a central nervous system pathogenesis of the Argyll Robertson syndrome,[3-4, 9, 12, 16, 23-26, 37, 41-42, 49-51, 54, 58-61] it has seemed worthwhile to report these findings.

The slitlamp biomicroscope has been used previously to study Argyll Robertson pupils. McGrath[39] employed the biomicroscope to observe the early and late changes in the iris that accompany the Argyll Robertson syndrome. Lowenstein and others[34,35] used it to study the initial changes in pupillary responses in patients who ultimately developed an Argyll Robertson pupil. Lowenstein[34] also reported the effects of cocaine on the light response in a fully developed Argyll Robertson pupil.

The biomicroscope was chosen for the present study when I accidentally found, during a routine slitlamp examination of a tabetic patient, that there was one miotic, light-rigid pupil, while the other was larger and retained a slow reaction to light. This was especially notable since the hospital record mentioned bilateral Argyll Robertson pupils. The diagnosis of bilaterality was based on a gross examination of pupillary size and reactions. Therefore, it seemed logical to conclude that the biomicroscope is a valuable aid in studying the Argyll Robertson pupil.

Observations in the present study confirm the work of Lowenstein and of McGrath. These observations also direct attention to a high incidence of unilateral, miotic, light-rigid pupils in a series of 135 syphilitic patients.

The criteria used in the diagnosis of Argyll Robertson pupil in the present study are identical with those described by Argyll Robertson in 1869.[1] These criteria are: (1) The pupil is small, (2) the pupil does not respond to light no matter how bright, (3) the pupil has a brisk response to convergence-accommodation, (4) the pupil dilates imperfectly to atropine, (5) the retina is sensitive to light.

The following case report will illustrate how the biomicroscope method was discovered and used in the present study.

* From the Department of Ophthalmology, Northwestern University School of Medicine, Chicago, Illinois.

### Case report

In 1949, a 38-year-old Negro came to the University of Chicago Eye Clinic because of reduced vision that had partially incapacitated him for two years. When he first noticed the failing vision, he had consulted a physician who treated him with 16 daily injections of penicillin. When the visual impairment continued to progress for two years, the patient sought our advice. He was aware of having had a penile lesion at least 15 years ago and difficulty in walking for three years.

The significant eye findings in 1949 were: vision, R.E., 20/70; L.E., 20/50; both hampered by the pinhole. Both pupils were small and irregular; the right, 2.0 mm., the left, 3.5 mm. in diameter. Neither pupil reacted to light directly or consensually; both reacted well to accommodation.

Under the routine slitlamp examination, however, the larger left pupil did react when the beam was shifted from a limbal position to an intrapupillary position. The consensual reaction was also present. The smaller right pupil did not react. When the pupils were dilated with scopolamine for cycloplegic refraction, the maximal pupil diameter was 6.5 mm. and the marginal irregularities were more marked than during miosis. The refractive error was negligible.

The fundus had a pale disc with slight increase in cupping, narrow arteries, and normal fundus color. The visual fields showed a general peripheral constriction with sharp slope and a small central scotoma to a 2/1,000 mm. white target.

Serologic and spinal-fluid tests for syphilis were positive. A diagnosis of Argyll Robertson pupil, right eye, syphilitic optic atrophy, bilateral, and probable tabes dorsalis was made.

The patient was sent to the Syphilis Clinic for treatment of his systemic disease; there the diagnosis of tabes was confirmed but a diagnosis of bilateral Argyll Robertson pupils was made.

One year later the patient returned to the Eye Clinic for removal of a foreign body embedded in the right cornea. Four-percent cocaine instillation, for analgesia, produced dilatation of the right pupil from 2.0 mm. to 3.5 mm. Under the slitlamp, this dilated pupil now reacted to the beam. The left pupil still had its constriction response to light. Three hours later, the right pupil was reduced to 2.5 mm. and reacted to the slitlamp beam rarely.

Two weeks later, the pupils had the same status as on the first examination one year before; that is, the right pupil was 2.0 mm. wide without a light response; the left pupil 3.5 mm. with a light reaction both direct and consensual.

Since bilateral Argyll Robertson pupils had been diagnosed by the Syphilis Clinic, despite our slitlamp diagnosis of a unilateral one, it seemed logical to suppose that any statistics regarding the incidence of this disease could not be relied on if perusal of histories alone were used.

Biomicroscopy had been used by Lowenstein[38] to study incipient Argyll Robertson pupils and by McGrath[39] and Koby[30] to study the pathology of the iris. No one, however, has reported using this method to study the unilaterality of Argyll Robertson pupils. Moreover, since Argyll Robertson had specified in his original report[2] that the pupils were inactive to light, no matter how bright, the slitlamp beam was considered to be a reasonable stimulus.

### Procedure

The study was conducted on 135 consecutive patients, seen for treatment or follow-up tests, in the Northwestern University Syphilis Clinic during three winter months in 1951; of these 135 patients, 46 had neurosyphilis.

Preliminary pupillary examinations were done on all the patients in a dimly lighted room with a flashlight beam. The patients whose pupils did not respond to this stimula-

tion or whose pupil diameter was less than 3.0 mm. were selected for slitlamp biomicroscope study; all of them had neurosyphilis.

The pupillary margin of each iris was examined under the biomicroscope while the slitlamp beam was shifted from a near-limbus position on the sclera to an intrapupillary position. A millimeter rule under the pupil allowed measurements of the pupillary diameter before and after the pupil responded to light incident on the retina. The patients were directed to look at an object six feet away during this examination. Then, with the light in the pupillary area, they looked at the forehead of the examiner to elicit the accommodation-convergence reaction. Finally, an unexpected pinprick on the back of the hand was used to determine the pupillary response to pain.

The criteria for a diagnosis of Argyll Robertson pupil were taken from the original descriptions by Douglas Argyll Robertson in 1869.[1,2] This diagnosis was made if the pupils were less than 2.5 mm. in diameter and did not react to light or pain but did react briskly to accommodation. Imperfect dilatation to bella donna, as originally described,[1,2] was not sought. In all the patients, the retinas were sensitive to light.

The eyes that met these requirements were cocainized with one drop of four-percent cocaine instilled into the conjunctival sac and then the pupillary reactions were tested under the biomicroscope at 15-minute intervals. If no pupillary light response was present after two hours, the eye was noted as a zero reactor.

## Findings

Out of the 135 patients on whom preliminary pupil examinations were done, 26 patients had no gross pupillary reaction to light in one or both eyes; of these, 14 had, in addition, no pupillary response to accommodation in the light-rigid iris. Therefore, these patients were eliminated from the study. Essential data from the clinic records and from the slitlamp biomicroscopic examination of the remaining 12 patients follow and are summarized in Table 1.

## Case reports

### Case 3

W. F., aged 59 years, first came to the clinic August 16, 1950, because of a whirling feeling in her head. Findings of mental degeneration, bilateral Argyll Robertson pupils, positive blood and spinal-fluid tests for syphilis, and colloidal gold curve of 443,211,000 led to the diagnosis of taboparesis. She was treated with penicillin and bismuth.

Vision was: R.E., 20/25; L.E., 20/30.

Pupil and iris findings under the biomicroscope in October, 1951, were: R.E., diameter 2.5 mm., slow constriction to light, active constriction to accommodation, good dilatation to pain, irregular

TABLE 1

Data on patients studied

| Case | Miosis | | Pupil Reaction to Light | | Pupil Reaction to Pain | |
|---|---|---|---|---|---|---|
| | R | L | R | L | R | L |
| 1 | + | 0 | 0 | + | 0 | + |
| 3 | + | + | + | 0 | + | 0 |
| 5 | 0 | + | 0 | 0 | + | 0 |
| 6 | + | 0 | 0 | + | 0 | + |
| 7 | + | 0 | 0 | + | 0 | + |
| 10 | + | 0 | 0 | 0 | 0 | + |
| 12 | 0 | + | + | 0 | + | 0 |
| 13 | 0 | + | + | 0 | + | 0 |
| 14 | + | 0 | 0 | + | 0 | + |
| 15 | + | 0 | 0 | 0 | 0 | + |
| 20 | 0 | + | + | 0 | + | 0 |
| 25 | + | 0 | 0 | + | 0 | + |
| 26 | + | 0 | 0 | + | 0 | + |

pupil margin, fine radial striations in a blue iris; after cocaine, diameter 3.5, constricts to light. L.E., diameter 1.5 mm., no constriction to light, no dilatation to pain, active constriction to accommodation, pupil margin irregular, no collarette, fine radial striations in a blue iris; after cocaine, diameter 2.5, constricts to light.

Pupil diagnosis: Unilateral Argyll Robertson pupil, left eye.

CASE 5

C. F., aged 47 years, first came to the clinic May 9, 1949, because of inability to walk with the right leg and pains in both legs. Findings of absent knee and ankle jerks, ataxia, bilateral Argyll Robertson pupil, positive spinal-fluid test for syphilis, and colloidal gold curve of 1,123,100,000 led to diagnosis of tabes. Treatment was with mapharsen and penicillin.

Corrected vision was R.E., 20/20; L.E., 20/20.

Pupil and iris findings under the biomicroscope in October, 1951, were: R.E., diameter 4.0 mm., no constriction to light or accommodation, slight dilatation to pain, pupil margin irregular, no gross abnormalities in brown iris; after cocaine, diameter 4.0 mm., no constriction to light. L.E., diameter 2.5 mm., no constriction to light, no dilatation to pain, active constriction to accommodation, irregular pupil margin, no collarette, fine radial striations in brown iris; after cocaine, diameter 4.0, does constrict to light.

Pupil diagnosis: Argyll Robertson pupil, left eye.

CASE 6

C. M., aged 34 years, first came to the clinic November 20, 1950, complaining of pains in abdomen, worse at night, and inability to see as well as usual. Findings of absent knee and ankle jerks, reduced visual fields, bilateral Argyll Robertson pupils, positive blood and spinal-fluid tests for syphilis, and colloidal gold curve of 2,234,410,000 led to a diagnosis of optic atrophy and tabes dorsalis. Treatment was with penicillin and bismuth.

Corrected vision was: R.E., 20/70; L.E., 20/50.

Pupil and iris findings under the biomicroscope in October, 1951, were: R.E., diameter 2.5 mm., no constriction to light, no dilatation to pain, active constriction to accommodation, irregular pupillary margin, no collarette, transilluminates at the 10-o'clock and at the 4-o'clock positions, fine radial striations in brown iris; after cocaine, diameter 3.5 mm., does constrict to light. L.E., diameter 3.0 mm., slow constriction to light, slow constriction to accommodation, good dilatation to pain, round pupil margin, no gross iris abnormalities; after cocaine, diameter 4.5 mm., constricts to light.

Pupil diagnosis: Unilateral Argyll Robertson pupil, right eye.

CASE 7

W. M., aged 61 years, first came to the clinic July 21, 1939, because of advice of employer who said patient was dropping too many things. Findings of ataxia, poor orientation to environment, absent ankle jerks, unilateral Argyll Robertson pupils, positive blood and spinal-fluid tests for syphilis, and colloidal gold curve of 4,433,210,000 led to diagnosis of taboparesis. Treatment was with bismuth and neoarsphenamine.

Vision was: R.E., 20/50; L.E., 20/40.

Pupil and iris findings under the biomicroscope October, 1951, were: R.E., diameter 1.5 mm., no constriction to light, no dilatations to pain, active constriction to accommodation, irregular pupil margin, brown iris transilluminates at the 3- and at 7-o'clock positions, fine radial striations; after cocaine, diameter 3.5 mm., does constrict to light. L.E., diameter 4.5 mm., dilates to light, immobile to accommodation and to pain, margin round, no iris abnormalities; after cocaine, diameter 6.0 mm., no constriction to light.

Pupil diagnosis: Unilateral Argyll Robertson pupil, right eye.

CASE 10

C. F., aged 52 years, first came to the clinic January 27, 1939, because of pain in the eyes and photophobia following a burn. Pain was present in the knees and legs. Findings of positive serologic and spinal-fluid tests for syphilis, and colloidal gold curve of 00-1-0-0-0 led to diagnosis of latent neurosyphilis. Treatment was with mapharsen and bismuth until January 5, 1945.

Corrected vision was: R.E., 20/20; L.E., 20/20.

Pupil and iris findings under the biomicroscope in November, 1951, were: R.E., diameter 2.0 mm., no constriction to light, good constriction to accommodation, no dilatation to pain, irregular pupil margin, no collarette, fine radial striations; after cocaine, diameter 2.0 mm., no constriction to light. L.E., diameter 3.5 mm., immobile to light and accommodation, slight dilatation to pain, round pupillary margin, no iris abnormalities; after cocaine, diameter 5.0 mm., no constriction to light.

Pupil diagnosis: Unilateral Argyll Robertson pupil, right eye.

CASE 12

W. M., aged 49 years, first came to the clinic March 4, 1937, because of sores on his feet. Findings of indolent ulcers on the soles of his feet, bilateral Argyll Robertson pupils, positive blood and spinal-fluid tests for syphilis, and colloidal gold curve of 4,443,321,000 led to a diagnosis of taboparesis. He was treated with bismuth and neoarsphenamine until February 9, 1948.

Pupil and iris findings under the biomicroscope in November, 1951, were: R.E., diameter 3.5 mm., good constriction to light and accommodation, good dilatation to pain, irregular pupil margin, collarette absent on lateral half of brown iris; after cocaine, diameter 3.5 mm., has constriction to light. L.E., diameter 2.5 mm., no reaction to light or pain, active constriction to accommodation, pupil margin irregular, no collarette on brown iris, fine radial stria-

tions; after cocaine, diameter 2.5 mm., no constriction to light. Vision was: R.E., 20/25; L.E., 20/25.

Pupil diagnosis: Unilateral Argyll Robertson pupil, left eye.

CASE 13

C. M., aged 31 years, first came to the clinic September 15, 1951, complaining of bad digestion and pains in the abdomen after eating. Findings of positive serologic and spinal-fluid tests for syphilis, bilateral Argyll Robertson pupils, absent ankle jerks, ataxia, early optic atrophy, and colloidal gold curve of 0,233,410,000 led to a diagnosis of tabes dorsalis. Treatment was with penicillin and bismuth.

Vision was: R.E., 20/30; L.E., 20/30.

Pupil and iris findings under the biomicroscope in November, 1951, were: R.E., diameter 3.5 mm., constricts slowly to light, well to accommodation, dilates to pain, margin irregular, collarette absent from the 10- to 12-o'clock positions and from the 4- to 5-o'clock positions; after cocaine, diameter 5.0 mm., constricts to light. L.E., diameter 2.5 mm., no constriction to light, no dilatation to pain, margin irregular, no collarette, transilluminates from the 4- to 6-o'clock positions, fine radial striations; after cocaine, diameter 3.5 mm., constricts to light.

Pupil diagnosis: Unilateral Argyll Robertson pupil, left eye.

CASE 14

W. F., aged 37 years, first came to the clinic March 4, 1947, complaining of trouble seeing and getting around at night. Findings of partial optic atrophy, absent knee and ankle jerks, positive blood and spinal-fluid tests for syphilis, and colloidal gold curve of 2,223,210,000 led to diagnosis of neurosyphilis. Treatment was with mapharsen, penicillin, and malaria.

Vision was: R.E., 20/70; L.E., 20/50.

Pupil and iris findings under the biomicroscope in November, 1951, were: R.E., diameter 2.0 mm., no constriction to light, no dilatation to pain, active constriction to accommodation, irregular margin, transilluminates, fine radial striations; after cocaine; diameter 2.0 mm., no constriction to light. L.E., diameter 3.0 mm., constricts to light and accommodation, dilates to pain, irregular pupil margin, fine radial striations from the 7- to the 9-o'clock positions on blue iris; after cocaine, diameter 4.0 mm., constricts to light.

Pupil diagnosis: Unilateral Argyll Robertson pupil, right eye.

CASE 15

C. F., aged 57 years, first came to the clinic April 10, 1946, complaining of aching legs and back. Findings of absent ankle jerks, positive spinal-fluid tests for syphilis, colloidal gold curve of 0,022,211,110, and bilateral Argyll Robertson pupils led to a diagnosis of neurosyphilis. Treatment was with mapharsen and bismuth.

Vision was: R.E., 20/30; L.E., 20/30.

Pupil and iris findings under the biomicroscope November, 1951, were: R.E., diameter 2.0 mm., inactive to light and pain, constricts to accommodation, margin irregular, transilluminates at the 7-o'clock position, fine radial striations; after cocaine, diameter 3.5 mm., constricts to light. L.E., diameter 3.0 mm., very slightly inactive to light, constricts to accommodation, dilates to pain, margin irregular, collarette absent; after cocaine, diameter 3.5 mm., slight reaction to light.

Pupil diagnosis: Unilateral Argyll Robertson pupil, right eye.

CASE 20

W. M., aged 42 years, first came to the clinic June 6, 1943, because of a fractured leg. Findings of bilateral Argyll Robertson pupils, positive blood and spinal-fluid tests for syphilis, and colloidal gold curve 0,010,100,000 led to a diagnosis of latent neurosyphilis. Treatment was with mapharsen, bismuth, and penicillin.

Vision was: R.E., 20/20; L.E., 20/20.

Pupil and iris findings under the biomicroscope December, 1951, were: R.E., diameter 4.0 mm., slow constriction to light, active constriction to accommodation, dilates to pain, margin irregular, fine radial striations, absent collarette from the 2- to 5-o'clock positions; after cocaine, diameter 6.0 mm., constricts to light. L.E., diameter 2.0 mm., immobile to light and pain, active constriction to accommodation, margin irregular, transilluminates at the 6-o'clock position, fine radial striations, collarette absent from the 12- to 5-o'clock positions; after cocaine, diameter 2.0 mm., no reaction to light.

Pupil diagnosis: Unilateral Argyll Robertson pupil, left eye.

CASE 25

W. M., aged 47 years, first came to the clinic May 11, 1944, because of trouble in walking and failing vision. Findings of absent knee and ankle jerks, arthritic changes in the right knee, optic atrophy, positive spinal-fluid tests for syphilis, and colloidal gold curve 3,332,110,000 led to diagnosis of neurosyphilis. Treatment was with mapharsen and bismuth until June 12, 1948.

Vision was: R.E., 20/70; L.E., 20/70.

Pupil and iris findings under the biomicroscope December, 1951 were: R.E., diameter 2.0 mm., immobile to pain and to light, active constriction to accommodation, irregular pupil margin, no collarette, fine radial striations; after cocaine, diameter 3.5 mm., constricts to light. L.E., diameter 3.0 mm., slow constriction to light, slow dilatation to pain, active constriction to accommodation, margin irregular, fine radial striations from the 4- to 7-o'clock positions, after cocaine, diameter 4.5 mm., constricts to light.

Pupil diagnosis: Unilateral Argyll Robertson pupil, right eye.

CASE 26

W. F., aged 56 years, first came to the clinic

October 15, 1942, because of intractable backache and headaches. Findings of ataxia, absent ankle and knee jerks, positive Romberg, absent vibratory sensation on legs, positive blood and spinal-fluid tests for syphilis, and colloidal gold curve 4,443,210,000 led to diagnosis of taboparesis. Treatment was with mapharsen and bismuth and malaria.

Vision was: R.E., 20/30; L.E., 20/25.

Pupil and iris findings under the biomicroscope December, 1951, were: R.E., diameter 2.5 mm., no constriction to light, no dilatation to pain, constricts to accommodation, irregular pupil margin, collarette absent, transilluminates at the 2-, 4-, and 8-o'clock positions, fine radial striations; after cocaine, diameter 4.0 mm., constricts to light. L.E., diameter 3.5 mm., constricts to light slowly, dilates to pain, constricts to accommodation, margin irregular, transilluminates at the 3-o'clock position; after cocaine, diameter 4.5 mm., constricts to light.

Pupil diagnosis: Unilateral Argyll Robertson pupil, right eye.

## Discussion

In these 13 cases, the patients had one pupil that met the criteria for a diagnosis of an Argyll Robertson syndrome and one pupil that did not. In none of them was the complete Argyll Robertson triad of miosis, light-rigidity, and absent pain response present in both eyes. In every instance, the pupil of smaller diameter was the light-rigid one.

It should be noted that in no case were the characteristics of the contralateral pupil normal. In all these, the light response was diminished in amplitude or had a long latent period. In all the patients, however, this abnormal but reactive pupil was larger than 2.5 mm. and a response to light was present either with or without an accompanying accommodation reaction so that a diagnosis of Argyll Robertson pupil could not be made.

All these 13 patients, therefore, met the conditions specified in Jessop's[29] definition of a unilateral Argyll Robertson pupil—namely, a pupil which is miotic and unable to react to light incident on the ipsilateral or contralateral retina although the pupil responds well to accommodation-convergence while the other pupil is larger and responds in the normal direction to all stimuli or responds abnormally but not in the manner described by Argyll Robertson in 1869.

The further study of these patients for iris abnormalities and for responses after cocainization had been suggested by the unusual findings in the first case reported herein. The abnormalities of the pupillary margin of the iris and of the iris stroma were noted and the light response of the pupil after cocainization was observed. These findings are summarized in Table 2.

The iris of every eye found to have an Argyll Robertson pupil was distinctly abnormal. The abnormalities were the same as those described by Grainger-Stewart,[19] Dupuy-Dutemps,[14] Koby,[30] Lemierre,[33] Fillippi-Gabardi,[15] McGrath, [20] Schaeffer,[52] Lowry,[36] and Orlando and Gambino.[46]

The crypts and collarette were obliterated

TABLE 2

Abnormalities of pupillary margin of iris and of iris stroma

| Case | Pupil Diameter (mm.) | Iris Abnormalities | | | | After Cocainization | |
|---|---|---|---|---|---|---|---|
| | | Irregular Margin | Absence of Collarette | Trans-illumination | Abnormal Striations | Pupil Diam. mm. | Light Reaction |
| 1 | 2.5 | + | + | + | + | 4.0 | + |
| 3 | 1.5 | + | + | | + | 2.5 | + |
| 5 | 2.5 | + | + | | + | 4.0 | + |
| 6 | 2.5 | + | + | + | + | 3.5 | + |
| 7 | 1.5 | + | | + | + | 3.5 | + |
| 10 | 2.0 | + | + | | + | 2.0 | 0 |
| 12 | 2.5 | + | + | | + | 2.5 | 0 |
| 13 | 2.5 | | + | + | + | 3.5 | + |
| 14 | 2.0 | + | | + | + | 2.0 | 0 |
| 15 | 2.0 | + | | + | + | 3.5 | + |
| 20 | 2.0 | + | | + | + | 2.0 | 0 |
| 25 | 2.0 | + | + | | + | 3.5 | + |
| 26 | 2.5 | + | + | + | + | 4.0 | + |

Fig. 1 (Apter). *Case 3* (M. P.). Left eye. Iris shows abnormal striations, miosis, irregular margin, and collarette absent between the 9- and 2-o'clock positions.

and fine radial striations were seen (figs. 1 and 2). In eight cases, the iris could be transilluminated.

It is notable that every iris capable of dilating to cocainization was also capable of constricting following this dilatation when light was incident on the ipsilateral or contralateral retina. Lowenstein[34,35] and Myerson and Thau[45] have previously reported the ability of the Argyll Robertson pupil to respond in this way to drugs like cocaine and benzedrine which produce mydriasis without paralysis of pupillary reflexes.

On the other hand, it is not logical to presume that the only reason a true Argyll Robertson pupil does not respond to light is because of its miosis. In other conditions of miosis, such as early acute Horner's syndrome,[24] congenital miosis,[23] senility, or pilocarpine-induced miosis, the pupil retains its light response despite the miosis. The cocaine effect was reversible and the pupils were again miotic and light-rigid in 24 hours. This effect was consistently reproducible.

The reaction to light which follows dilatation with cocaine merely suggests (but does not prove) that interference with the light reflex is not the significant factor in the pathologic process of this syndrome. The study does describe the incidence of a fully developed Argyll Robertson syndrome in patients observed under the biomicroscope.

Ten of the cases in the present study were called bilateral Argyll Robertson pupils in the hospital record on the basis of gross examinations. Yet, these 10 patients had a true unilateral Argyll Robertson syndrome. In all the patients both eyes were involved and were in the process of becoming an Argyll Robertson syndrome, but in all cases one eye had become fully developed before the other so that one pupil was miotic and light-rigid while the other was not.

The study also supports the contention of McGrath,[39] Langworthy,[31] and Myerson[45] who have separately maintained that the Argyll Robertson pupil does not lose its light reaction until the miosis is marked. None of these patients had the characteristics described by Ford and others[17] of unilateral Argyll Robertson pupils following third-nerve regeneration.

The present findings suggest that the uni-

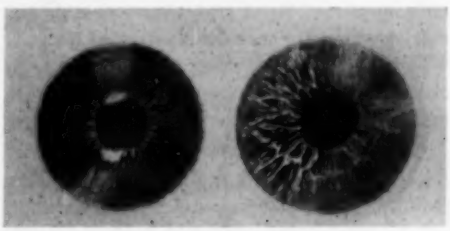


Fig. 2-A (Apter). Drawings of partially abnormal irises in which the abnormal sectors did not respond to light incident on the retina, whereas the more normal sectors did respond. Diagnosis was incipient Argyll Robertson syndrome.

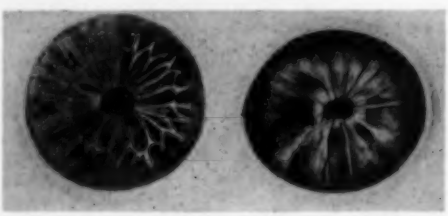


Fig. 2-B (Apter). Drawings of abnormal irises in patients with an Argyll Robertson syndrome. Reproduced with permission from McGrath.[39]

lateral Argyll Robertson pupil should not be regarded as a rare condition. Therefore, a theory that attempts to localize the lesion responsible for this syndrome is not satisfactory unless it can explain all the signs originally ascribed to this syndrome, whether it occurs in one or both eyes. Another requisite of a theory of pathogenesis is that it be consistent with pathologic findings.

The central nervous system theory of pathogenesis[4, 6, 22, 25, 37–38, 41, 49, 51, 54, 58–61] fails to satisfy both of these requisites. Light-rigidity alone has been explained in this theory by a lesion in the afferent pupillary light pathway close to the first synapse in the pretectal region. This lesion, however, does not produce a small pupil. Moreover, a lesion in that location is seldom found in neurosyphilis. On the other hand, the miosis is ascribed to a separate lesion that has not yet been allocated.

None of the central nervous system lesions proposed thus far could explain a unilateral, miotic, light-rigid pupil. Furthermore, neurologists agree that there is no known central nervous system lesion that could possibly produce a unilateral Argyll Robertson syndrome. Therefore, those who support a theory of a central nervous system pathogenesis of the Argyll Robertson pupil are forced to postulate two lesions—one to explain the miosis and another to explain the light-rigidity.

Since each sign—the miosis and the light-rigidity—may occur separately in many diseases, while the combination of miosis and light-rigidity occurs only in syphilis, it would appear worthwhile to search for a single lesion to explain the Argyll Robertson syndrome. The manner in which the present clinical data support the postulate that the single lesion may be in the iris itself will be the subject of a future report.

Before these theories can be well substantiated, however, it is essential that knowledge be obtained concerning the precise pathologic process responsible for the gross abnormalities of the iris. In this connection, microscopic study of irises from patients who had the complete Argyll Robertson syndrome might prove fruitful in directing attention toward a lesion intrinsic in the iris that could explain all the characteristics of the Argyll Robertson pupil and of the unilateral syndrome as well.

## Summary

1. The slitlamp biomicroscope provides a very sensitive, accurate technique for studying the Argyll Robertson syndrome of miosis, absence of pupillary light response, and overactive pupillary accommodation reaction.

2. With the use of the biomicroscope, unilateral Argyll Robertson pupils were found in 12 of 46 patients with neurosyphilis. None had the Argyll Robertson syndrome in both eyes.

3. Gross abnormalities of the iris, such as transillumination, absence of collarette, and fine radial striations were associated with each miotic, light-rigid pupil.

4. Cocaine produced dilatation of most of these miotic pupils and, following this mydriasis, the pupils were capable of constricting when light was incident on the ipsilateral or contralateral retina.

5. These findings are incompatible with the theory of a central nervous system pathogenesis of the Argyll Robertson pupil.

*7316 South Paxton Street (49).*

## References

1. Argyll Robertson, D.: On an interesting series of eye symptoms in a case of spinal disease, with remarks on the action of belladonna on the iris. Edin. M. J., **14**:496, 1869.

2. ———: Four cases of spinal miosis with remarks on the action of light on the pupil. Edin. M. J., **15**:487-493, 1869.

3. Behr, C.: Zur Physiologie und Pathologie des Licht Reflexes der Pupille. Arch. f. Ophth., **86**:468-513, 1913.

4. ———: Ueber Metalues des Auges. Ztschr. f. Augenh., **60**:319-334, 1926.

5. Biyadoux, A., and Gourevitch, F.: Les troubles du pré-Argyll Robertson (de la reflexometrie pupillaire). Presse Med., **45**:550, 1937.

6. Boeke, J.: Problems of Nervous Anatomy. London, Oxford University Press, 1940.

7. Casper, L.: Beobacklungen uber einseitige reflectorische Pupillenstarre. Arch. f. Augenh., **54**:63, 1896.

8. ———: Ein Fall von einseitiger, reflectorischer Pupillenstarre. Arch. f. Pract. Augenh., **32**:291-293, 1896.

9. Chavany, J. A., and Lemoine, P.: Syndrome de la calotte pédonculaire et de la région des tubercles quadrijumeaux. Le signe d'Argyll Robertson symptôme focal et non etiologique. Bull. Med. Paris, **48**:92-94, 1934.

10. Clark, C. P.: Eye changes observed in paretic patients after treatment with malaria. Am. J. Ophth., **13**:946-955, 1930.

11. Derum, F. X.: The pupil in tabes, paresis and syphilis. J. Nerv. & Ment. Dis., **33**:718-720, 1906.

12. Dreyfus, D. A.: Un nouveau cas d'Argyll Robertson unilateral par tumeur recidivante du sinus maxillaire si propageant vers l'orbite. Rev. d'otoneuro-ocul. **5**:200-203, 1927.

13. Duke-Elder, W. S.: Textbook of Ophthalmology. London, Henry Kimpton, 1949.

14. Dupuy-Dutemps, L.: Sur une forme spéciale d'atrophie de l'iris au cours du tabes et de la paralysie générale, ses rapports avec l'irregularité et les troubles réflexes de la pupille. Ann. d'ocul., **134**:190-210, 1905.

15. Fillippi-Gabardi, E.: Le alterazioni dell iride nella sindrome di Argyll Robertson. Riv. oto-neuro-oftal., **10**:737-752, 1933.

16. Foerster, O.: Zur Pupillarinnervation. Deutsche Ztschr. f. Nervenh., **106**:313, 1938.

17. Ford, F. R., Walsh, F. B., and King, A.: Clinical observations on the pupillary phenomena resulting from regeneration of the third nerve with especial reference to the Argyll Robertson Pupil. Bull. Johns Hopkins Hosp., **68**:300-318, 1941.

18. Globus, J. H.: Tumors of quadrigeminate plate; clinico-anatomic study of 7 cases. Arch. Ophth., **5**:418-444, 1931.

19. Grainger-Stewart, T.: The eye symptoms in locomotor ataxia. Brain, **2**:181-190, 1879.

20. Harris, A. J., Hodes, R., and Magoun, H. W.: Afferent path of the pupillodilator reflex in the cat. J. Neurophysiol., **7**:231-243, 1944.

21. Harris, W.: The fibers of the pupillary reflex and the Argyll Robertson pupil. Arch. Neurol. & Psychiat., **34**:1195-1203, 1935.

22. Holmes, G.: Partial iridoplegia associated with symptoms of other diseases of the nervous system. Tr. Ophth. Soc. U. Kingdom., **51**:209-224, 1932.

23. Holth, S., and Berner, O.: Congenital miosis or pinhole pupils owing to developmental faults of the dilator muscle. Brit. J. Ophth., **7**:401-419, 1923.

24. Horner, F.: Ueber eine Form von Ptosis. Klin. Monatsbl. f. Augenh., **7**:193-198, 1869.

25. Igersheimer, L.: Syphilis und Auge. Berlin, Springer, 1918.

26. Ingvar, S.: Ueber einseitiges Argyll Robertson. Acta, Med. Scandinav., **34**:180-181 (Suppl.) 1930.

27. Inman, W. S.: The nonluetic Argyll Robertson pupil. Brit. M. J., **2**:1179-1180, 1925.

28. Jaeger, E.: Einseitige pupillenstarre. Klin. Monatsbl. f. Augenh., **97**:658-661, 1936.

29. Jessop, W. M. H.: On the consensual pupillary light reflex in cases exhibiting the Argyll Robertson symptom in one eye. Tr. Ophth. Soc. U. Kingdom, **11**:249, 1890-1891.

30. Koby, J.: Microscopy of the Living Eye. Philadelphia, Blakiston, 1930, p. 213.

31. Langworthy, O. R., and Ortega, L.: The Iris. Medicine, **22**:287-361, 1943.

32. Lawson, F. H.: Nonluetic Argyll Robertson Pupil. Brit. M. J., **2**:1180-1181, 1925.

33. Lemierre, A., Garcin, R., and Laplane, R.: Sur une forme cachectique de myotonie atrophique (maladie de Steinert). Edentation totale. Ebauche du signe d'Argyll Robertson. Rev. Neurol., **39**:898-905, 1932.

34. Lowenstein, O.: Die Storungen des Lichtreflexes der Pupille bei den luetischen Erkrankungen des Zentralnervensystems Beitrage zur Fruhdiagnostik de Lues Nervosa. Basle, Benno Schwabe et Cie., 1935.

35. Lowenstein, O., and Friedman, E. D.: Adie's syndrome (Pupillotoni pseudotabes). Arch. Ophth., **28**:1042-1068, 1942.

36. Lowry, J. T.: Iris changes in syphilis: Further observations. Southwestern Med., **21**:290-293, 296-297, 1941.

37. Lutz, A.: The light pupillary reflex, its path and its abolition, called immobility of the pupil to the light reflex and report of a case of unilateral Argyll Robertson pupil in which consensual reaction existed. Arch. Ophth., **47**:266-292, 1918.

38. Maltrass, F. J.: Cases illustrating the etiology of the Argyll Robertson pupil. J. Neurol. & Psychopath., **4**:162-166, 1923.

39. McGrath, W. M.: Observations upon abnormalities of the pupils and iris in tabes dorsalis, general paralysis, and taboparesis, with consideration of their bearing upon the pathogenesis of Argyll Robertson pupils. J. Ment. Sci., **78**:363-373, 1932.

40. Menninger, W. C.: Unilateral Argyll Robertson pupil, with presence of consensual reactions in both eyes; report of a case. J. Nerv. & Ment. Dis., **63**:58-60, 1926.

41. Merritt, H. H., and Moore, M.: The Argyll Robertson pupil. An anatomic and physiologic explanation of the phenomenon with a survey of its occurrence in neurosyphilis. Arch. Neurol. & Psychiat., **30**:357-373, 1933.

42. Moore, R. F.: The nonluetic Argyll Robertson pupil. Brit. M. J., **2**:843-844, 1925.

43. Moreau, R., Bertrand-Fontaine, Mme., and Garcia, R.: Signe d'Argyll Robertson par abcès de la calotte pédonculaire au cours d'une meningo-éncephalite suppurée à pneumocoques. Rev. Neurol., **2**:117-121, 1930.

44. Morelli, J. B., and Izola, A.: El signo de Argyll Robertson unilateral. Rev. Med. d'Uruguary, **29**:253-259, 1926.

45. Myerson, A., and Thau, W.: Human autonomic pharmacology, effect of benzedrine sulfate on the Argyll Robertson pupil. Arch. Neurol. & Psychiat., **39**:780-788, 1938.

46. Orlando, R., and Gambino, L. R.: Investigationes sabre la fisiopatogenia del signo de Argyll Robertson, la curva del oro coloidal en el humor acuoss de los paraliticos generales. Rev. Neurol. Buenos Aires, **5**:38-43, 1940.

47. Parifique, and Bussy, J.: Syndrome de l'apex orbitaire. Signe d'Argyll Robertson seule sequelie d'attiente du moteur oculaire commun. Bull. Soc. d'opht. de Paris, 1932, No. 8, pp. 621-624.

48. Parks-Weber, F.: Argyll Robertson pupils with mydriasis. Proc. Roy. Soc. Med., Children's Sect., **16**:68, 1923.

49. Ranson, S. W., and Magoun, H. W.: Central path of the light reflex; study of the effect of lesions. Arch. Ophth., **13**:701-811, 1935.

50. ———: Central path of the pupillo-constrictor reflex in response to light. Arch. Neurol. & Psychiat., **30**:1193, 1933.

51. Scala, N. P., and Spiegel, E. A.: The pupillary reactions in combined lesions of the posterior commissure and of the pupillodilator tracts. A.M.A. Sect. Ophth., 1935, pp. 184-208.

52. Schaeffer, H., and Herigot de Treigny: Le signe d'Argyll Robertson; Ses rapports avec l'atrophie irienne. Rev. d'oto-neuro-ocul., **14**:609-628, 1936.

53. Shapira, M. F., and Crage, F. M.: Pupillary variability in 108 syphilitic patients, Am. J. Ophth., **19**:891-893, 1936.

54. Spiegel, E. A.: Argyll Robertson Pupil. Urol. & Cuban Rev., **45**:428-432, 1941.

55. Tanzi, E.: Singulare contegno delle pupille in un cas iniziale di paralisis progressiv. Riv. di Pat. nerv. e Ment., **4**:385-390, 1899.

56. Thomson, H. C.: Argyll Robertson pupil caused by mesencephalic tumor. Practitioner, **110**:289-290, 1923.

57. Tille, H.: Signe d'Argyll Robertson unilatéral avec mydriase par lésion peripherique probable dans un cas de tabes incipiens. Bull. Soc. d'opht. de Paris, 1933, pp. 629-632.

58. Walsh, F. B.: Clinical Neuro-Ophthalmology. Baltimore, Williams & Wilkins Company, 1947, pp. 193-195.

59. Wilson, S. A. K.: Problems in Neurology: The Argyll Robertson Pupil. J. Neurol. Psychopath., **11**:1, 1921.

60. Wilson, S. A. K., and Gerstle, M.: Argyll Robertson pupils in mesencephalic tumors. Arch. Neurol. & Psychiat., **22**:9-18, 1929.

61. Wilson, S. A. K.: The Argyll Robertson pupil. In: Modern Problems in Neurology. New York, W. Wood & Co., 1929, pp. 332-363.

## Ophthalmic Miniature

. . . Your eye is the lamp of your body; when your eye is sound, your whole body is full of light; but when it is not sound, your body is full of darkness. . . .

*The Gospel According to Luke,*
Chapter 11, verse 34.

# BILATERAL HOMONYMOUS HEMIANOPSIA*

FREDERIC M. REESE, M.D.
*Baltimore, Maryland*

Bilateral loss of vision of sudden onset has many causes and occurs under a variety of circumstances. This paper is concerned with bilateral loss of vision resulting exclusively or principally from posteriorly situated lesions, that is, from lesions affecting the upper visual pathways. Blindness from such lesions may occur as a result of anoxia and following ventriculography and cerebral arteriography. It may result from hypertensive encephalopathy, cerebral hemorrhage, thrombosis, and spasm in the cerebral arteries, air embolism, direct injury to the skull, and from blood-transfusion reactions.

Attention is called to a recent paper by Behrman[1] on blindness of sudden onset, both temporary and permanent.

The importance of anoxia as a cause of "blackout" or transient blindness or as a cause of extensive and permanent damage to the central nervous system has become more widely recognized since Haldane's investigations were published in 1922.[2] Haldane pointed out that oxygen-want not only stopped the engine for want of fuel but, if severe enough, caused "total ruin of what we took to be machinery."

Anoxia may occur under a number of conditions. It has been classified as follows: (Barcroft's original classification[3]):

1. Stagnant, in which there is a reduction of the blood flow in the tissues, as in shock due to vasomotor collapse, embolus, or thrombosis.

2. Anoxic, in which there is interference with the passage of oxygen from the pulmonary alveolar spaces into the capillaries as in pneumonia, plugging of the trachea or bronchi with foreign matter, drowning, or inhalation of gases of low oxygen content.

3. Anemic, in which the oxygen-carrying capacity of the blood is reduced, due to insufficient hemoglobin or modification of the hemoglobin, as in poisoning from carbon monoxide or from the venom of certain snakes.

Peters and van Slyke[4] added another type: Histotoxic, in which the tissue cells are unable to use the oxygen made available to them by the blood, as in alcohol, narcotic, or cyanide poisoning.

Extensive investigations, both experimental[5–8] and clinical,[9–11] have shown that, with anoxic anoxia of moderate severity even for a matter of minutes, irreversible changes may occur in the central nervous system resulting in cortical blindness and even in death. Some parts of the brain, notably the occipital cortex, are more susceptible to anoxia than are others (Burdick, Lisa, and Elias;[9] Douglas;[10] and Hoff, Grenell, and Fulton[11].) The transient effects of anoxia have been discussed by Behrman. Serious and irreversible destruction resulting from prolonged and severe anoxic anoxia is illustrated by three of the four case histories now summarized.

## CASE HISTORIES

### CASE 1

An infant was born while the mother was in an oxygen tent with pneumonia. A previous attack of pneumonia had also occurred during the pregnancy. Severe permanent visual loss resulted in the child.

This white infant was seen when she was about one year of age. At birth cyanosis was severe, but good color appeared rapidly and remained. The mother believed the child did not see even at a few months of age. The child was slow to stand.

At about one year of age there was no evidence of any vision. The optic discs were extremely pale and possibly slightly recessed. The pupils reacted well to light. The tendon

* From the Wilmer Institute of The Johns Hopkins University and Hospital.

reflexes were brisk but if any spasticity was present it was slight.

Loss of vision was believed to be mainly cortical because of the persistence of the pupillary reactions.

CASE 2

Permanent loss of vision resulted after laryngeal spasm with apnea for three minutes during bronchoscopy under sodium-pentothal anesthesia. The patient had syphilis and there was a history of epilepsy from the age of 20 years.

This Negro, age 36 years, was seen about eight months after the onset of his loss of vision. He gave a history of "epileptic spells" for 15 years; these were often brought on by bouts of coughing. The diagnosis was hyperplastic tracheobronchitis. An attempt to bronchoscope the patient under local anesthesia was unsuccessful. Bronchoscopy was then repeated under sodium-pentothal anesthesia. Severe laryngeal spasm occurred with cessation of breathing for at least two or three minutes.

After the anesthesia was withdrawn, the patient vegetated for the next two or three weeks and was completely blind. Eight months later he was able to take fluids successfully by mouth for the first time and he was able to talk thickly but coherently. One month later vision was limited to light perception. The fundi were normal and the pupils reacted properly to light.

CASE 3

Permanent loss of vision following severe postoperative hemorrhage after tonsillectomy with obstruction of the trachea by a blood clot for three to five minutes. This was followed by blindness, stupor, Cheyne-Stokes respiration, muscular twitchings, and convulsions with the slightest stimulation. Blindness persisted.

This white man, aged 33 years, was seen about two and a half months after the anesthetic accident. He had had tonsillitis and peritonsillar abscesses with repeated incision and drainage. He was given a nitrous-oxide induction and ether anesthesia for removal of his tonsils and adenoids. The operation lasted only about 20 minutes and the patient was returned to his bed in good condition.

Shortly afterward, he developed a profuse hemorrhage from the tonsillar fossae. He was given morphine. About two and a half hours after the operation, he became cyanotic and his radial pulse was not palpable. A minute or two later his respirations stopped and cyanosis increased markedly. Artificial respiration, oxygen, and suction were used. A clot seven by one and a half inches was removed from the patient's throat three or four minutes later. Artificial respiration was necessary for another half hour.

On the second day, after the operation the patient was stuporous and showed convulsive movements with stimulation. On the fifth day, he was able to talk. He complained that he could not see. Retinal edema and papilledema were reported to be present.

On the seventh day, he still was unable to see light. The fundi appeared normal at this time. His pupils reacted to light on the twelfth day and light perception was present. Two and a half years after the operation the patient could see only hand movements although his fundi and pupillary reactions were normal.

CASE 4

Temporary total loss of vision following vomiting and aspiration of food particles during delivery of a normal baby at term under sodium-pentothal anesthesia. Decerebrate rigidity developed and later the same day the patient responded to painful stimuli only. After two days, marked improvement of vision was evident and it ultimately returned to normal. The visual fields and fundi were also normal.

This white woman, aged 38 years, was seen in The Johns Hopkins Hospital during her illness. During delivery, the patient vomited and aspirated food particles while under the anesthetic. She was unable to breathe and ex-

hibited intense cyanosis for about 15 minutes. A tracheotomy was done and a good pink color returned at once.

Two and a half hours later the patient showed decerebrate rigidity. Seven hours after the respiratory difficulty she was still rigid but responded to painful stimuli. The next day she was able to swallow water.

The pupils reacted well to light stimulus and on the second day vision had definitely improved. On the third day, a slight sensory aphasia was noted and there was marked impairment of fine movements. The reflexes were hyperactive.

On the ninth day, vision was about 2/200 in each eye. The ocular movements, pupillary reactions, and fundi were normal. On the 11th day, vision was about 2/100 in the right eye and 2/70 in the left. Visual field studies showed a left lower homonymous quadrant defect for four degrees, red and blue test objects.

After seven weeks, vision had returned to 20/15 in each eye and the visual fields and fundi were normal. An examination 18 months after the original trouble showed no abnormality.

### Comment

Ford, Walsh, and Jarvis[12] reported a case of blindness following respiratory failure in a patient under nitrous-oxide and ether anesthesia. Artificial respiration was required for a few minutes during the operative procedure—repair of a hernia. When the anesthetic was withdrawn, the patient remained in a coma for 36 hours, during which time several generalized convulsions occurred and there was some elevation of the patient's temperature. Later there was marked loss of cerebration, and cerebellar ataxia, apraxia, and aphasia were noted. These abnormalities gradually cleared and the visual fields improved with a residual homonymous scotoma extending to the fixation point in each left lower quadrant. Central acuity remained quite low.

These five cases are examples of the serious damage to the central nervous system, including the upper visual pathways, which may follow anoxic anoxia due to respiratory difficulties. Although the number of cases is small, it is obvious that, in general, anoxic anoxia of considerable duration produces severe changes. Case 3 exemplifies the extreme sensitivity of the visual apparatus to anoxia since the visual defect was the sole residue of the anesthetic mishap. It is probably reasonable to state that, with severe anoxic anoxia, there is likely to be permanent blindness if the patient lives.

Courville[13] described 13 cases of damage to the central nervous system following nitrous-oxide induced anesthesias, in eight of which sudden respiratory failure occurred and, in four, a feeble or imperceptible pulse was noted. In four of these patients, no symptoms occurred during the anesthesia but the patients remained in a deep coma when the anesthesia was withdrawn. Mental deterioration was pointed out as the most common sequela.

Löwenberg and others[14] reported four more cases, three of which terminated fatally. Histologic changes in the central nervous system consistent with the findings in experimentally produced anoxia in animals[7,8] were present in Courville's and Löwenberg's cases.

## Visual loss following ventriculography

### Case 5

A white man, aged 50 years, lost all vision following ventriculography. Vision ultimately improved to 20/20−1 in the right eye and 20/20−2 in the left eye with a residual right lower quadrantanopsia.

The patient was admitted to the hospital with a history of severe headaches which could be reproduced by histamine injections. Desensitization to histamine failed to bring relief. Blood pressure determinations showed pressures of around 190/120 mm. Hg, and there was a well-developed arteriosclerotic hypertensive type of retinitis with exudates and hemorrhages in each eye.

The right upper eyelid drooped slightly

and the right pupil was definitely smaller than the left one. Both pupils reacted normally to light stimulus. The ocular movements were entirely normal.

Pressure over the right internal carotid artery (Matas test) for 10 minutes was well tolerated and in the beginning seemed to relieve the headaches. This vessel was ligated in the neck with marked relief of the headaches for a few days. They returned, however, and were more severe than before the ligation.

Eleven days after the ligation an air injection was made into the cerebral ventricles. The right lateral ventricle filled promptly and appeared to be normal. The left lateral and third ventricle could not be demonstrated. Several attempts to reach the left ventricle were unsuccessful.

Following the air injection, there was complete loss of vision with "hysterical reactions difficult to interpret." The patient was irrational for several days and seemed to be quite blind. Some slight return of vision was noted eight days after the air injection was made. The headaches continued. Visual acuity improved rapidly and on the 11th day it was said to be good. There was a residual right homonymous hemianopsia at this time. The headaches, right partial ptosis, and miosis persisted.

The patient was not seen again for several years. Recently he stated that the severe headaches continued for about two years and then gradually diminished so that he had been free of them for the past four years. His vision had remained normal. Recently his corrected vision was 20/20−1 in the right eye, and 20/20−2 in the left eye.

The right upper eyelid still drooped slightly and the right pupil was smaller than the left one. Both reacted normally to light stimulus.

The retinal arteries showed definite changes characteristic of arteriosclerosis but there were no exudates or hemorrhages in either fundus. The nerveheads were normal. Visual field studies showed a right lower homonymous quadrantanopsia for one-degree white.

### Case 6

A white man, aged 45 years, exhibited a right homonymous hemianopsia as a result of a craniopharyngioma. After ventriculography there was temporary loss of vision in the left eye with pronounced subsequent recovery.

Loss of vision to the right had been noted by the patient two years before this examination. A neurologic examination was entirely negative except for an incongruous, incomplete, right homonymous hemianopsia with reduction of vision to 20/100 in the right eye and 20/40 in the left eye.

A Diodrast injection into the left carotid artery indicated a small suprasellar mass. Three days later an air injection was done which was followed by complete loss of vision in the left eye. Two days later light perception was present and one day later perception of hand movements in the temporal field.

Seven days after the injection of air, vision had improved to the ability to count fingers and on the 12th day it was 5/200.

The next day a craniotomy was done on the right side and a large (two cm. in diameter) cyst, extending into the sella and causing great tension on the right optic nerve, was removed. Microscopic examination showed this to be a craniopharyngioma.

Six days later vision in the right eye was 20/200 and in the left eye 20/100. Two years and nine months after the operation vision was 20/40 in each eye (bilateral macular degeneration), and the visual fields were markedly improved on both sides of the vertical meridian.

### Case 7

A white boy, aged seven years, with a history of frontal headaches, nausea, and vomiting, a right sixth nerve palsy, cracked-pot sound over the skull, and severe papilledema with 20/40 vision in each eye, lost all vision

following ventriculography. Improvement started within 36 hours and vision and visual fields ultimately returned to normal.

On admission, the patient's vision was 20/40 in each eye and there was a severe degree of papilledema in each eye with exudates and hemorrhages. The visual fields could not be charted due to lack of co-operation.

An air injection was done which revealed a lesion on the right side of the brain. A craniotomy was done on the same day. A tumor was not found and the skull was closed without replacing the bone flap.

Following the operation the patient complained bitterly and repeatedly of not being able to see. Twenty-four hours later, however, he was able to see the doctor's hands moving before his face. He could also tell colors to some degree. The patient was discharged from the hospital three days later.

Four and a half years later the child's vision was entirely normal and his health excellent. He has been attending school regularly and doing very well.

### Comment

When ventriculography is performed, it is always possible that insertion of the needle into the posterior horn of the ventricle is responsible for the visual field defect or that hemorrhage has developed in the occipital lobe.

These three cases of visual loss following ventriculography were characterized by immediate total loss of vision in both eyes in two cases and total loss of vision in one eye in the third. Marked improvement occurred in all three. Study of these three cases suggests that visual loss following ventriculography is temporary. This is in conformity with many published cases of such visual loss.

### Air embolism

The various effects of air embolism on the nervous system are well known. Embolism has been observed in (1) caisson disease and in (2) therapeutic and accidental introduction of air into the blood stream. This has been observed in (a) operations on the chest wall, (b) artificial pneumothorax, (c) injection of air into the sinuses, (d) a knee-chest position in women recently delivered, and (e) accidental wounding of the jugular vein in the neck. The amount of air necessary to cause symptoms is not known.

Air embolism frequently causes pain in the head, dizziness, fear of impending death, a rapid pulse, shallow and labored breathing, and generalized or localized convulsions. Blindness sometimes results.

Blindness may be an early symptom lasting from a few minutes to several days. The pupils may be constricted or dilated. Paralysis of the ocular muscles has been reported. Homonymous quadrantic and hemianopic defects have also been reported. Since there are no examples of blindness from air embolism in this series of cases, I have drawn from the description of two such cases by Walsh.[15]

### Cases from literature

A patient had the first stage of a thoracoplasty with resection of the first, second, and third ribs, and extrafascial apicolysis followed by pneumothorax. An injection of 120 cc. of air under 220 mm. of water pressure was made and immediately following this there was excruciating pain in the head. In a few minutes nausea and vomiting occurred and the patient was observed to be blind. The pupils were constricted.

For the next three days the patient showed fear of impending death, restlessness, coma, and convulsions. By the fifth day, however, he was greatly improved and able to feed himself. The ocular movements were normal at all times. Macular edema was observed on one occasion. On the fourth day vision had improved to the perception of hand movements, on the fifth day it was 20/200 and by the ninth day 20/15 in each eye.

The next case was also one of pulmonary tuberculosis in which a left-sided extra-

pleural pneumolysis followed by injection of 500 cc. of air was done. Two hours later sudden loss of vision occurred with motor paralysis of all extremities, disorientation, fear of death, nausea, and vomiting. The pupils were dilated and fixed to light. Retinal pallor was observed, but disappeared four hours later.

Paralysis of the right side disappeared the next day. The left arm could be moved on the sixth day. On the fourth day there was light perception in each eye and the pupils reacted to light stimulus. Within two weeks vision had improved to 20/20 in each eye. However, a bronchocutaneous fistula developed, signs of sepsis followed, and the patient died in three weeks.

From a study of these two cases it seems that although loss of vision following air embolism may be complete, recovery may be confidently anticipated if the patient survives for a day or two. Behrman[1] described a similar case with complete loss of vision and recovery after four days.

Monocular blindness following injection of paraffin into the orbit for cosmetic reasons is mentioned here because it has been reported in the literature along with fat embolism. Leber[16] reported eight such cases and Uhthoff[17] one case. Obstruction of the central retinal artery was observed in all of these cases.

Vascular obstruction by fat embolism is so frequently fatal that blindness is not described. However, cases of involvement of the retinal vessels are reported without serious loss of vision and with complete recovery.

Fritz and Hogan[18] described two such cases in young men with extensive bone injuries. One of these patients recovered and the other died. Evans[19] described a similar case with retinal changes and rapid recovery. I have been unable to find any cases of cortical blindness due to fat embolism in the literature. It is interesting that fat embolism, if extensive enough to cause blindness, causes death; whereas, blindness with air embolism is followed by complete recovery if the patient survives.

### Blood supply of visual cortex

A brief discussion of the vessels supplying the visual cortex, the posterior cerebral arteries, is indicated at this point. The paired terminal vessels are given off from the basilar artery in the interpeduncular space just above the emerging oculomotor nerves which pass between these vessels and the superior cerebellar arteries. The trochlear nerves also lie between these two sets of vessels. Shortly after their origin, the posterior cerebral arteries receive blood from the internal carotid arteries by way of the posterior communicating vessels.

Each posterior cerebral artery first passes forward, then backward and upward around the cerebral peduncle, below the splenium of the corpus callosum, and then to the visual cortex of the occipital lobe. Shellshear[20] demonstrated beautifully the area supplied by the calcarine artery by injecting a dye into the posterior cerebral artery and then sectioning the occipital lobe. He demonstrated that the area colored by the dye was the same as outlined by the line of Gennari in the visual cortex.

In its early course, branches are given off to the red nucleus, the corpora quadrigemina, the geniculate bodies, and the posterior part of the optic thalamus, including the pulvinar. Most of the visuopsychic, as well as the visuoperceptive area, is supplied by this vessel.

As a result of the ample blood supply from the internal carotid arteries by way of the posterior communicating arteries, thrombosis or closure of the basilar artery otherwise does not usually result in visual field changes. "Sparing of the macula" in occipital vascular lesions[21] probably results from the blood supply made possible by the anastomoses between the terminal branches of the posterior cerebral and middle cerebral arteries in this

area. Förster[22] was probably the first to point out this double blood supply in 1890.

Attention is also drawn to the position of the posterior cerebral arteries at their commencement for here they are particularly vulnerable to pressure either from prolapse of the temporal lobe through the tentorium or elevation upward of the cerebellum.

During part of their course they come close to the free edge of the tentorium. A supratentorial tumor which produces herniation of the uncus through the incisura tentorium is likely to compress the posterior cerebral artery of that side.

A not uncommon post-mortem finding is cortical necrosis of both occipital lobes of the type produced by such arterial obstruction.[22] This finding has recently been observed in a case of bilateral homonymous hemianopsia not associated with tumor or trauma. In this case, which was not observed by me, a thrombus extended from the anterior end of the basilar artery into both posterior cerebral arteries to beyond the junction with the posterior communicating arteries.

It would be anticipated that with involvement of the basilar or posterior cerebral arteries that diplopia, ptosis, or other evidence of brain-stem damage might be present. Such symptoms have been observed in very few of the cases reported here.

Bilateral homonymous hemianopsia probably arises most frequently as a result of widespread vascular disease. Its development has been observed in individuals suffering from vascular hypertension in which instance it may be associated with hypertensive encephalopathy. However, bilateral homonymous hemianopsia often occurs when the blood pressure has not been unduly elevated. In these cases it would seem that thrombosis is the etiologic factor.

What the role of arterial spasm is in cases of bilateral homonymous hemianopsia is not clear. However, in those cases with extremely rapid recovery it is easy to visualize spasm as the inciting cause. On the basis of hemianopsia often being associated with migraine and in some instances being permanent, the question arises as to whether or not bilateral homonymous hemianopsia might originate from migraine. Certainly there is no proof of this.

### Bilateral homonymous hemianopsia with hypertensive encephalography

#### Case 8

A white woman, aged 53 years, with a history of hypertension for many years, showed complete loss of vision in the left eye and only light perception in the right eye about one year after an iridectomy on the left eye for acute glaucoma. The glaucoma was not considered to be responsible for the loss of vision which did not improve.

A year before my examination, the patient had an acute attack of glaucoma in the left eye. An iridectomy controlled the tension and the patient had not been seen again until pain was experienced in the right eye three days before examination. Pilocarpine had been prescribed for the right eye after the operation as a precautionary measure. Furmethide was prescribed for the right eye at once and examination soon afterward showed the intraocular pressure in this eye to be 15 mm. Hg.

In spite of this low tension and a normal tension in the operated left eye, there was only questionable light perception in the right eye and no light perception in the left eye. The right pupil was noted to be small and reactive and the pupillary margins in the left eye did not move with light stimulus. Corneal sensitivity and the ocular movements were normal.

A vitreous hemorrhage below the disc and a hard white exudate temporal to the macula were noted in the right eye. The retinal arteries showed a severe grade of arteriosclerosis and several other small hemorrhages were observed in both eyes. The left disc was extremely pale with some suggestion of glaucoma. There was a moth-eaten appearance in the macula. The blood pressure taken

on several occasions was found to be around 210/105 mm. Hg.

CASE 9

An 83-year-old surgeon experienced sudden total loss of vision several hours after having done a long, tedious, and tiring operation. He experienced marked exhaustion and his blood pressure was found to be acutely elevated (systolic of 300 mm. Hg). Total blindness lasted 24 hours and was accompanied by delirium, subjective visual sensations, vertigo, diplopia, amnesia, and incontinence. Recovery was ultimately complete.

The patient had always enjoyed good health until his "attack." He had been a vigorous, driving, hard-working individual. His blood pressure had not been previously unusually high. After completing his operation, he felt exhausted and retired early.

About half an hour later he experienced a "sudden, severe dizziness as if I'll die." Everything seemed to spin around from right to left and his head felt enormous. He had the illusion of pushing his son to the ceiling for a minute or two.

According to the patient's wife he was totally blind for the next 24 hours. Diplopia was then experienced and persisted off and on for several weeks. Four days after the onset of the blindness, he was delirious and had all sorts of imaginary ideas. He complained of not being able to taste his food. He was ataxic in the dark and lost control of his bladder and bowels. He recovered and performed a radical mastectomy six and a half months after the original episode.

Examination at about this time showed loss of hearing for high tones and some loss of vibratory sense in the legs attributed to the patient's advanced age. There was no clear evidence of a focal cerebral vascular lesion. Vision was 20/30+4 in each eye and the visual fields were normal. The nerveheads appeared to be normal and the retinal arteries showed generalized narrowing and caliber variation. There was a single splinter-shaped hemorrhage in the left fundus. The only other finding brought out at this time was that the patient had been a "mild diabetic for six or seven years."

BILATERAL HOMONYMOUS HEMIANOPSIA WITH VASCULAR DISEASE BUT WITHOUT UNDULY ELEVATED BLOOD PRESSURE

CASE 10

A 60-year-old white woman, with a history of a "stroke" three years previously, who had had a large colloid goiter removed eight years previously, followed by paralysis of the vocal cords and frequent attacks of tetany, experienced total blindness following a second "stroke." There was a return of the central fields only.

A huge colloid goiter weighing 375 gm. was removed in 1943 with relief of symptoms of hyperthyroidism and congestive heart failure. This operation, however, was followed by paralysis of both vocal cords which necessitated a tracheotomy with a permanent indwelling tube. Symptoms of severe hypoparathyroidism also developed which required calcium therapy. The patient's blood pressure was around 125/80 mm. Hg.

The first "stroke" occurred in 1948 and resulted in paralysis of the left arm and leg. The patient was not aware of any visual loss at this time. The second "stroke" occurred 26 months later and was followed by total blindness. The patient was not sure how long this lasted. Central vision, however, returned and remained.

An examination of the patient's eyes was made about 10 months after the second "stroke." At this time the ocular movements were normal, the corneas clear, and the pupils equal and reactive.

The fundus of the right eye was not well seen because of lens opacities. The left fundus showed a granular appearance in the macular area believed to be a degenerative process. The retinal arteries showed signs of moderately severe arteriosclerosis. The nervehead in this eye appeared to be normal. No exudates or hemorrhages were seen.

Vision was 20/70 in the right eye and

4/200 in the left eye with correction. The visual fields were limited to a few degrees about the fixation point in each eye.

Dr. Frank R. Ford examined the patient on the same day and commented as follows: "There are signs of a left hemiparesis with spasticity and increased tendon reflexes. No sensory loss. The visual fields consist of a few degrees around the fixation point. Lenticular opacities are present. The pupils react to light and there are no gross changes in the nerveheads. The patient probably has bilateral softenings involving the occipital cortex or optic radiations. Possibly she had a left hemianopsia with the first stroke and a right one with the second. I suspect, however, she had a bilateral homonymous hemianopsia with the second stroke as she believes. I have seen this repeatedly and return of central vision is characteristic."

CASE 11

A white man, aged 51 years, experienced sudden loss of vision while at dinner. A small field extending about 10 degrees to the right of the fixation point only remained in each eye. This was accompanied by paresthesias, weakness of the left leg, and mild clumsiness of the right hand. The blood pressure was 150/86 mm. Hg.

The patient was examined in The Johns Hopkins Hospital about five months after the original incident. He gave a history of having had "leaky heart valves for 30 years probably following rheumatic fever." He had also been treated for syphilis but it was thought that tests may have been a "false positive." He had had no other symptoms, however, preceding the present illness except for mild chronic headaches.

Following the onset of visual loss, the patient had trouble holding a cigarette in his right hand. He managed to finish dinner, however, and retired to his room where his lack of vision became more apparent when he tried to read. He remained in bed for the next three weeks and was unable to read during this time. Improvement of vision was then noted, especially to the right. He was able to return to work after four or five weeks. About a month later he felt drowsy and exhausted. He returned home and supposedly had a generalized convulsion with loss of consciousness lasting one or two minutes. He did not regain consciousness for three hours. He then slept for 12 hours.

Signs of aortic and mitral valvular disease were noted, as well as aortic changes believed to be due to arteriosclerosis. The neurologic examination at this time was negative except for the visual fields which showed a left homonymous hemianopsia with some slight clearing in the left lower fields. Vision was 20/20 in each eye.

The fundi were normal except for very moderate arteriosclerosis of the retinal arteries. Electroencephalograms gave entirely negative results with the eyes closed. With the eyes open there was definite evidence of a lesion in the right occipital lobe extending to the left.

CASE 12

A white man, aged 68 years, experienced sudden loss of vision while driving his car. Flashes of light were observed in the beginning and severe headaches followed and lasted for 24 hours.

The patient was seen here for the first time 21 months after the attack of visual loss. Although he still complained of blurring of vision, he was aware of marked improvement in both eyes. Careful questioning revealed that he could see only in a small central field for several months. Previous to the attack he had been generally well and had had no visual difficulties.

Examinations revealed moderate arteriosclerosis with low blood pressure, but nothing else of consequence. The eyes were entirely normal externally. The right fundus was entirely normal except for a large "drusen" above the macula. The left fundus showed a few "drusen" in the upper part of the macular region. The retinal arteries showed only moderate arteriosclerotic

changes. Vision in the right eye was 20/30−1 and in the left eye 20/30+2. The visual fields were almost full in all meridians.

CASE 13

A white woman, aged 58 years, with a history of auricular fibrillation, experienced sudden loss of vision while dressing. There was momentary improvement after a few minutes and then severe loss which lasted for two weeks with some clearing near the fixation point subsequently.

The patient was examined here for the first time about six weeks after the visual loss. The history revealed nothing of consequence except that auricular fibrillation had been noted on one occasion. The blood pressure had never been unduly elevated and was found to be 150/82 mm. Hg. The patient had apparently had no other symptoms, such as headaches, dizziness, subjective visual sensations, or hallucinations associated with the visual loss. Over the six weeks' period little if any improvement had been noted.

Examination showed the pupils to be equal and normally reactive and the ocular movements to be normal. Vision was about 1/15 in each eye and the field of vision was limited to a few degrees in the central portion of each left inferior quadrant. Optokinetic nystagmus could not be elicited.

In all probability this case exemplifies lodgement of an embolus or development of a thrombus at the junction of the basilar and posterior cerebral arteries with extension beyond the posterior communicating arteries.

CASE 14

A white man, aged 60 years, with a history of inadequately treated syphilis and moderate arteriosclerosis, experienced sudden severe loss of vision while driving his car. Improvement was noted first after eight days and finally reached 20/20 in the right eye and 20/30+3 in the left eye with a residual left homonymous hemianopsia.

The patient was seen first in The Johns Hopkins Hospital about 19 months after the loss of vision occurred. The patient gave a history of having been treated for syphilis 30 years before. He had been given 25 "arm shots" at that time. His serologic test for syphilis became negative but 19 months ago it was found to be positive again. He was treated this time with penicillin. The amount of the treatment was not known. The patient had had no other symptoms, visual or otherwise, before the present illness.

The sudden loss of vision occurred while the patient was driving his car. No other symptoms accompanied the visual loss which persisted and the patient was admitted to the hospital the next morning because of his unexplained blindness. He could see only light and had to be led about for eight days. Improvement was noted from this time on and was more pronounced in the beginning in the right eye.

Examination 19 months after the original episode showed a questionable bilateral ptosis and some inequality of the pupils with incomplete reactions to light stimulus. The left pupil was slightly larger than the right. The ocular movements were normal. The fundi were normal except for moderate arteriosclerosis of the retinal arteries and a questionable hemorrhage in the extreme upper portion of the left fundus was observed. The blood pressure was 170/60 mm. Hg. Visual acuity was 20/20 in the right eye and 20/30+2 in the left eye. Visual field studies showed a left homonymous hemianopsia. An examination 10 months later showed no significant change.

CASE 15

A white man, aged 63 years with moderately severe generalized and cerebral arteriosclerosis without hypertension and with mental deterioration, developed complete loss of vision. Complete dementia and finally death ensued.

The patient was admitted to The Johns Hopkins Hospital complaining of blurring of vision for three weeks. According to his

wife, he had been worried over mental deterioration for several months. He had recently been aware of scintillating scotomas and was extremely nervous and unable to sleep.

When admitted to the hospital on April 27, 1951, his blood pressure was 125/85 mm. Hg and there was evidence of moderate generalized and cerebral arteriosclerosis. The retinal arteries showed corresponding changes. The pupils reacted normally and the ocular movements were normal. There was a left homonymous hemianopsia.

Examination of the spinal fluid was entirely negative and ventriculograms showed nothing.

After several days the patient began to deteriorate mentally and showed evidence of more visual loss. By May 18, 1951, he could not recognize a water pitcher or locate familiar objects in his room. He became markedly confused and disoriented.

From this time on his general condition gradually became worse. There were periods of severe agitation. Convulsions occurred and inability to empty his bladder developed. Opisthotonus was observed. He died on June 30, 1951. An autopsy was done.

The anatomic and neurosurgical microscopic notes follow:

*Anatomic diagnosis.* Cerebral atrophy. Multiple encephalomalacias. History of ventriculograms. Bilateral burr holes, cranium. Slight recent subdural hemorrhage, left occipital lobe.

*Neurosurgical microscopic note.* Sections from the cerebral cortex show widespread foci of softening. These are small, except for one in the visual radiation of the right occipital lobe measuring 3.0 by 5.0 mm. These are confined to the white matter of the cerebral cortex and are characterized by cavitation, myelin destruction, and peripheral infiltration by round cells many of which are laden with hemosiderin.

The frontal and both occipital sections show more of this than the temporal lobes which are only minimally involved.

Small, fresh perivascular hemorrhages are numerous in the frontal and both occipital lobes and are present in all sections including those of the brain stem. In addition, perivascular infiltration by one or two layers of round cells is widespread. The ganglion cells of the frontal cortex show considerable increase in number and size and many of the neuronal elements remaining are shrunken. The process is equally marked in the left occipital lobe although the right occipital lobe is greatly spared. The temporal lobe shows areas in which this condition is prominent existing side-by-side with normal appearing regions. Accompanying this change is marked proliferation in glial elements.

The blood vessels, apart from the hemorrhages and infiltration mentioned above, appear normal.

*Diagnosis.* Multiple encephalomalacia. Cerebral atrophy.

### Discussion of Cases 8 through 15

In these cases there was severe visual loss in individuals between the ages of 51 and 83 years and with various degrees of arteriosclerosis but, for the most part, with only moderate arteriosclerosis and not unduly elevated blood pressure (eight of 10 cases).

In one case (8), the blood pressure had been elevated for many years and there was a well-developed arteriosclerotic hypertensive type of retinitis with exudates and hemorrhages. In another case (9), the blood pressure had never been unduly elevated except during an attack of visual loss when the systolic pressure was found to be around 300 mm. Hg. In the remaining cases the retinal arteries showed only moderately severe arteriosclerotic changes.

In Cases 11, 12, 13, and 14 the loss of vision was known to have been sudden and to represent the first and only symptom except for "flashes of light" and headaches for 24 hours in one case.

In Case 11, there was a history of rheumatic fever and evidence of aortic and mitral valvular disease. In Case 13, there was

a history of auricular fibrillation and in Case 14 inadequately treated syphilis existed. In Case 10, total blindness was present after a second "apoplectic" attack in a 57-year-old woman with a blood pressure of 125/80 mm. Hg and signs of only moderate arteriosclerosis.

In Case 15, blindness occurred almost as an incidental symptom during the course of a rapidly progressive encephalomalacia with cerebral atrophy due presumably to arteriosclerosis. Microscopic studies made in this case showed widespread changes in the brain including the occipital lobes.

Study of these case histories shows that in Case 8 with long-standing hypertension, in Case 10 with two "strokes" but no increase in the blood pressure, and in Case 13 with a blood pressure of 150/82 mm. Hg, visual improvement was very limited or absent. In Case 9, however, with a temporary elevation of the blood pressure to 300 mm. Hg (systolic) and in Cases 11, 12, and 14 with average blood pressure levels, there was great improvement. This improvement was most complete in Case 9. In four cases, one with hypertension and three without, the neurologic examination was negative at all times except for the loss of vision. In one of these, Case 13, there was little or no improvement.

It seems clear then that bilateral homonymous hemianopsia may be associated with hypertensive encephalopathy but occurs more frequently when hypertension and signs of severe arteriosclerosis (including changes in the retinal arteries) are absent. Probably there is no correlation possible between the presence or absence of hypertension, the degree of arteriosclerosis, suddenness of onset, the amount of visual loss, or the degree of improvement.

### Blindness following blood-transfusion reaction

Walsh[24] described a case of permanent blindness resulting from cortical injury as a result of a transfusion reaction of hemolytic type in a boy with sickle-cell anemia. Convulsions and unconsciousness were observed in this case.

Blood was found in the spinal fluid and was believed to have resulted from a subarachnoid hemorrhage. The blood pressure was found to be temporarily severely elevated during the attack and marked spasm was observed in the retinal arteries.

Six months later vision was still limited to perception of light. The retinal arteries were approximately normal at this time.

### Bilateral homonymous hemianopsia following direct injury to head

#### Case 16

A young white woman, aged 17 years, experienced severe loss of vision after an injury to the head in an automobile accident eight months before being seen at The Johns Hopkins Hospital. There was no improvement in the visual fields after eight months' observation.

The patient was semicomatose for two and a half weeks following the injury. A neurologic examination made soon afterward was reported to have been negative. Several weeks later the reflexes were more brisk on the right and the left eye diverged about 20 degrees and could not converge normally. The fundi and pupillary reactions at this time were reported to be normal.

Examination eight months after the injury was entirely negative except for the visual field changes (remaining field of vision extended from the fixation point to 30 degrees in each left upper quadrant) and the external squint of the left eye. Vision was 20/20 in each eye. The fundi and pupillary reactions were normal. An electro-encephalogram was reported as follows: A low-voltage disorganized record with absence of alpha rhythm of the right occipital area.

It is believed that there was bilateral damage to the occipital lobes or optic radiations. It seems probable on a basis of the visual field changes (central fields only remaining) that the injury was occipital and likely resulted from interruption of the blood supply.

### Bilateral homonymous hemianopsia following cerebral arteriography

The patient, a Negress, aged 24 years, was given an injection of Diodrast into the right common carotid artery. During the injection, the left carotid artery was occluded by digital pressure. Total blindness developed almost immediately. Bare perception of light returned in each eye after an hour during which time a bilateral stellate ganglion block was performed. On the following day the right half of each field of vision regained some function and within 36 hours visual acuity and fields for both eyes were normal. There was not at any time an ophthalmoscopic picture of retinal arterial occlusion (courtesy of Dr. F. B. Walsh and Dr. G. W. Smith).

## Summary and Conclusions

Bilateral homonymous hemianopsia as observed in 20 cases has been described in this paper. These cases exemplify its occurrence in association with or due to anoxia, ventriculography, air embolism, vascular obstruction, blood-transfusion reaction, injury, and cerebral arteriography.

Although the symptomatology of these conditions is well known, with the exception of blindness following cerebral arteriography, it has been briefly restated with the idea that some new ideas might arise as a result of considering these cases together. The following conclusions seem to be justified and of importance.

1. It is clear that bilateral homonymous hemianopsia or cortical blindness occurs under a variety of circumstances.

2. Although it is well known that bilateral homonymous hemianopsia occurs frequently in association with hypertensive encephalopathy, in eight cases due presumably to vascular changes, and described here, hypertension was present in only two.

3. It is of interest that among these cases (vascular etiology) evidence of damage to the brain stem occurred definitely in only one, Case 9, and questionably in another, Case 14. Attention has been drawn to the vulnerability of the posterior cerebral arteries, yet blindness in the majority of these cases was due to involvement of these vessels peripheral to the branches supplying the brain stem.

4. On a basis of the cases described herein, certain generalizations regarding prognosis for return of vision seem to be valid. In blindness due to anoxia, the prognosis tends to be bad (three of four cases remained essentially blind). In the three cases of visual loss following ventriculography, recovery was complete in all cases. In two cases of blindness due to air embolism recovery was complete.

In individuals with loss of vision associated with hypertensive encephalopathy or vascular obstruction in the absence of hypertension, some degree of recovery was the rule. In the eight cases described here normal central vision returned in four. A residual left homonymous hemianopsia was present in two of these cases and some peripheral constriction in all quadrants persisted in a third.

Little or no improvement was observed in three cases (8, 13, 15) and central vision returning in one (10) was reduced by ocular changes. One of these patients (15) died.

In the single case of visual loss following a blood-transfusion reaction, improvement was negligible.

Return of central vision only was noted in the patient with visual loss following the head injury. This would seem to indicate that the loss of vision resulted from obstruction of the blood supply to the visual cortex.

5. A single case of bilateral homonymous hemianopsia occurring during the injection of Diodrast into one carotid artery with digital occlusion of the other carotid has been described. Complete recovery of vision and visual fields occurred within 36 hours.

*330 North Charles Street (1).*

## References


1. Behrman, S.: Amaurosis fugax et amaurosis fulminans. Arch. Ophth., **45**:458-467, 1951.
2. Haldane, J. S.: Respiration. New Haven, Conn., Yale University Press, 1922.
3. Barcroft, J.: Anoxemia. Lancet, **2**:485-489, 1920.
4. Peters, J. P., and van Slyke, D. D.: Quantitative Clinical Chemistry. Baltimore, Williams & Wilkins, 1931, ed. 1, v. 1, p. 579.
5. Grenell, R. G.: Central nervous system resistance: Effects of temporary arrest of cerebral circulation for periods of 2 to 10 minutes. J. Neuropath. & Exper. Neurol., **5**:131-154 (Apr.) 1946.
6. Weinberger, L. M., Gibbon, M. H., and Gibbon, J. H., Jr.: Temporary arrest of the circulation to the central nervous system: Pathological effects. Arch. Neurol. & Psychiat., **43**:961-986 (May) 1940.
7. Kabat, H., and Dennis, C.: Decerebration in the dog by complete temporary anemia of the brain. Proc. Soc. Exper. Biol. & Med., **38**:864-865 (June) 1938.
8. Gildea, E. F., and Cobb, S.: Effects of anemia on the cerebral cortex of the cat. Arch. Neurol. & Psychiat., **23**:876-903 (May) 1930.
9. Burdick, D. L., Lisa, J. R., and Elias, M. G.: Anoxia and the central nervous system: Review and case report. Anesthesiology, **10**:369-373 (July) 1949.
10. Douglas, S.: Cerebral changes related to anoxia, with report of a case. Canada M.A.J., **61**:123-129 (Aug.) 1949.
11. Hoff, E. C., Grenell, R. G., and Fulton, J. F.: Histopathology of the central nervous system after exposure to high altitudes, hypoglycemia, and other conditions associated with central anoxia. Medicine, **24**:161-217 (May) 1945.
12. Ford, F. R., Walsh, F. B., and Jarvis, J. A.: A case of extensive injury to the cerebral cortex following nitrous oxide-ether anaesthesia. Bull. Johns Hopkins Hosp., **61**:246-257 (Oct.) 1937.
13. Courville, C. B.: Asphyxia as a consequence of nitrous oxide anaesthesia. Medicine, **15**:129-245 (May) 1936.
14. Löwenberg, K., Waggoner, R. W., and Zbinden, T.: Destruction of the cerebral cortex following nitrous oxide-oxygen anaesthesia. Ann. Surg., **104**:801-810 (Nov.) 1936.
15. Walsh, F. B.: Clinical Neurophthalmology. Baltimore, Williams & Wilkins, 1947, pp. 1006-1008.
16. Leber, T.: Die Krankheinten der netshaut: Besondere arten von embolie der zentralarterie: paraffinembolic. Graefe-Saemisch. Hand. Augenh., **7**:278-280, 1915.
17. Uhthoff, W.: Injury to the eyes after paraffin injections for saddle-nose. Ophth. Record, **15**:115-117, 1906.
18. Fritz, M. H., and Hogan, M. J.: Fat embolism involving the human eye. Am. J. Ophth., **31**:527-534 (May) 1948.
19. Evans, J. J.: Cerebral fat embolism with recovery and involvement of the central retinal artery. Brit. J. Ophth., **24**:614-616 (Dec.) 1940.
20. Shellshear, J. L.: A contribution to our knowledge of the arterial supply of the cerebral cortex in man. Brain, **50**:236-253 (June) 1927.
21. Strong and Elwyn: Human Neuroanatomy. Baltimore, Williams & Wilkins, 1943, p. 375.
22. Förster, O.: Ueber rindenblindheit. Arch. f. Ophth., **36**:94-108, 1890.
23. Moore, M. T., and Stern, K.: Vascular lesions in the brain stem and occipital lobe occurring in association with brain tumors. Brain, **61**:70-98 (Mar.) 1938.
24. Walsh, F. B.: Clinical Neurophthalmology. Baltimore, Williams & Wilkins, 1947, p. 1045.


## Ophthalmic Miniature

Often as a child, so I remember, I dropped oil of olives into my eyes, when it displeased me to commit to memory the famous words of the dying Cato.

Persius.

# EXOTROPIA WITH BILATERAL ELEVATION IN ADDUCTION*

## PART I. DIAGNOSIS

MARTIN J. URIST, M.D.
*South Haven, Michigan*

It has been of great advantage to be able to reclassify many cases, formerly lumped in a heterogeneous diagnostic group as cases of intermittent exotropia, into a definite clinical entity, exotropia with bilateral elevation in adduction (Group III of horizontal squint with secondary vertical deviations).[1] By so classifying these cases their diagnosis and treatment have become more accurate.

Scobee[2] summarized the problem of classification when he stated:

"Intermittent exotropia is known by many other names because there is, unfortunately, no standard terminology in the field of dysfunctions of the oculorotary muscles. Some know intermittent exotropia as 'marked convergence insufficiency' and others call it 'divergence excess.' Some call it 'periodic exotropia' and others speak of 'exophoria-exotropia.' The condition is characterized by moderate to marked degrees of exophoria lapsing into frank exotropia with inattention, fatigue, or prolonged use of the eyes for close work."

Bair,[3] in an article entitled "Intermittent exotropia," expressed the more common teaching of today by dividing these cases into three groups (according to Duane):

1. *Divergence excess*—cover measurements greater for distance than for near, good near-point of convergence.

2. *Divergence-excess plus convergence insufficiency*—cover measurements greater for distance than for near, poor near-point of convergence.

3. *Convergence insufficiency*—cover measurements greater for near than for distance, poor near-point of convergence.

Many have realized the inadequacies of a classification based on the convergence near-point and cover measurements since these findings can be so variable. Scobee[4] stated that the amount of exotropia for distance with the cover test was more often greater at 20 feet than for near although the reverse was not at all uncommon. If these patients are occluded for 24 hours or are taught to relax convergence, the measurements will be greater for near than for distance. Scobee goes on to say:

"The amount of exotropia which may be elicited upon examination of the patient with intermittent exotropia is inversely proportional to the degree of nervous tension present in the patient at the time. The greater the nervous tension, the less exotropia there will be manifested on the cover test or on any other test for phoria or tropia. For this reason, it is better for the surgeon who is relatively inexperienced in operating in such cases not to see his patients after they have been admitted to the hospital before the operation. The patient is always excited and nervous, and under such conditions the deviation may disappear entirely so that it cannot even be elicited with the cover test." (See Case 1 herein.)

Sugar,[5] in discussing the treatment of intermittent exotropia, said:

"It should be kept in mind that the measurement of the deviation in intermittent exotropia is often not the actual total latent deviation present. I have found it important in using the cover test with these cases to do the alternate covering slowly and to be sure that the deviation first be overcorrected with prisms and then brought back to the neutral point. If this is done, one may find that a case which was considered of the divergence excess type actually has as much, or nearly as much, deviation for near as for distance."

* From the Department of Ophthalmology of the Illinois Eye and Ear Infirmary, University of Illinois College of Medicine, Chicago.

Burian[6] stated: "I believe it to be a fallacy to base the choice of operation on the behavior of convergence and divergence."

Chavasse[7] said: . . . "We are perhaps able to abandon the rather sterile, question-begging, and clinically unsatisfactory classification of cases into (a) convergence insufficiency, (b) divergence excess, and (c) mixed, which includes most cases."

Any satisfactory clinical classification should take the vertical deviations into consideration, as I have done, since these are present in most cases. White and Brown[8] found that 59.9 percent of "divergence excess" cases had a vertical imbalance. Costenbader[9] found a vertical imbalance present for distance but either not present at all or much less when the patient was fixing at 13 inches. Scobee[2] found that in a series of 25 cases, 11 had a vertical imbalance.

White[10] stated: "In a personal interview with Dr. F. W. Marlow, I stated my impression that in divergence excess 70 percent of cases were complicated by vertical phoria. He thought it higher and later sent statistics that showed 85 percent to be so complicated. More careful observations later proved to me that we both were too conservative."

These vertical deviations, until recently, were considered to be due to paretic vertical muscles. Actually they are secondary to the dissociation of the eyes caused by exotropia, as was discussed in a previous paper.[1] Scobee became convinced of this also, and a comparison of his discussion of the vertical deviations found in intermittent exotropia in 1949 with that in 1952 illustrates the change in his ideas. In 1949 Scobee[2] stated:

"Of the 25 cases of intermittent exotropia presented, 11 had a vertical component (meaning evidence of paresis of one or more of the vertically acting muscles)."

He then presented a table in which the following muscles were believed to be affected: both inferior recti—two cases; left superior oblique—two cases; right superior rectus—two cases; left superior rectus—one case; and one case each of both superior recti, the left inferior oblique, the right inferior rectus, and both right elevators.

In 1952 he[11, 12] stated: "It is not at all uncommon to find a small to moderate amount of vertical deviation on the cover test in patients with intermittent exotropia. The vertical deviation is most commonly an apparent hyperphoria of the non-dominant eye. Search for a paretic vertically acting muscle usually reveals nothing of significance. One is therefore forced to conclude that such a hyperphoria is functional rather than organic and probably has something in common with the entity of alternating sursumduction. . . . The fact that such hyperphoria frequently disappears completely following surgery on the horizontal muscles only, lends added support to the assumption that the hyperphoria was functional in origin."

The significant findings in my series of 102 cases of exotropia with bilateral elevation in adduction will be given in tabular form. These findings will be compared with those of other recent authors in "intermittent exotropia" where possible. The findings are tabulated for all the patients for whom they were recorded. The only cases omitted for each table were the ones in which the relevant findings were not taken or recorded.

In Table 1, the comparison of my findings with those of other authors is remarkable in that the results are so nearly the same. About

TABLE 1

AGE OF ONSET IN INTERMITTENT EXOTROPIA

| Author | No. of Cases | Birth | Under 5 Years | 5 Years or Older | Post-operative | Unknown |
|---|---|---|---|---|---|---|
| Scobee | 25 | 36% | 40% | 4% | 0 | 20% |
| Costenbader | 528 | 39% | 45% | 4.5% | 0 | 10.5% |
| Urist | 102 | 43% | 36% | 8% | 9% | 2.6% |

TABLE 2

REFRACTIVE ERRORS IN INTERMITTENT EXOTROPIA

| Author | No. of Cases | Hypermetropia | Myopia | Mixed Astigmatism |
|---|---|---|---|---|
| Cass | 14 | 86% | 0 | 14% |
| Bair | 114 | 87% | 13% | |
| Urist | 83 | 57% | 19% | 24% |

TABLE 3

CONVERGENCE NEAR-POINT IN INTERMITTENT EXOTROPIA

| Author | No. of cases | 60 mm. or Less | Greater than 60 mm. |
|---|---|---|---|
| Scobee | 25 | 52% | 48% |
| Bair | 126 | 59% | 41% |
| Urist | 86 | 55% | 45% |

40 percent of the cases had a history of onset at birth and about 80 percent below five years of age. These cases run contrary to the usual concept of the age of onset of exotropia as summarized by Duke-Elder,[18] who stated: "Divergent squints tend to develop slowly, usually between puberty and mid-life, and their habit is to increase gradually as age advances."

My findings concur with those of others (table 2) in that hypermetropia was the predominant error and that myopia was not statistically significant in the etiology of the exotropia.

Table 3 shows that a convergence near-point of less than 60 mm. was only a little more common than that of more than 60 mm. I found the convergence near-point quite variable. Many cases showed a remote near-point some days and a good near-point on other days. In some patients one examiner could elicit a good near-point while another could not.

Sometimes placing minus 3.0D. lenses before the eyes would change a remote near-point to a very close one. Other patients with a remote near-point would have a very good one when stimulated by the taking of photographs (Cases 1 and 2). From these variable findings one can reasonably conclude that a classification depending on the near-point of convergence would not be reliable.

As can be seen in Table 4, the deviation measured more for near than for distance in 60 percent of my cases while Scobee found the deviation measured more for near in 40 percent and the same for distance in 28 percent.

When the corneal reflex method of examination was used (table 5), however, the findings were much different. In 10 percent the exotropia was greater for distance than for near, in 22 percent the same distance and near and in 68 percent an exotropia was present for distance while the eyes were straight for near. In no case was the exotropia for near greater than for distance.

Re-examination of the findings in those cases (table 5) that had an exotropia for distance and straight eyes for near revealed that the prism cover measurements were greater for near than for distance in 55 percent. Stereopsis was found in 91 percent and in nine percent was not determined. This indicated that an exophoria was present for near that could be greater than the exotropia for distance as determined with prism measurements. These findings required further clarification.

The amount of exotropia for distance (Hirschberg) was compared with the amount

TABLE 4

COVER MEASUREMENTS IN INTERMITTENT EXOTROPIA

| Author | No. Cases | Distance More than Near | Near More than Distance | Distance Same as Near | Up More than Down | Down More than Up | Bilateral Vertical Deviation | Unilateral Vertical Deviation |
|---|---|---|---|---|---|---|---|---|
| Scobee | 25 | 32% | 40% | 28% | | | 8% | 36% |
| Urist | 86 | 40% | 60% | 0 | 93% | 7% | 68% | 13% |

TABLE 5

CORNEAL REFLEX METHOD OF MEASUREMENT—83 CASES

| XT Distance Greater than Near | XT Distance, Straight Near | XT Distance Same as Near | XT Distance Less than Near | XT Up Greater than Down | XT Up, Straight Down | XT Up Less than Down | Bilateral Elevation in Adduction |
|---|---|---|---|---|---|---|---|
| 10% | 68% | 22% | 0 | 29% | 71% | 0 | 73% |

found with the prism cover measurements in the same cases. It was found that the exotropia as determined with the corneal reflex method was nearly twice the amount as found with the prism measurements in the majority of the cases. The discrepancy between the two methods of examination might be explained as follows:

A large exotropia may be present for distance and especially when the child is daydreaming. This is one of the characteristics of the condition and usually the reason the parents bring the child to the physician. Examination by the corneal reflex method is purely objective and does not disturb the child too much and, by this method, it is usually possible to measure the actual amount of exotropia that is present for distance.

When the prism measurements are taken the child must pay attention to the fixation object. This excites him and stimulates convergence and fusion which makes the exotropia much smaller than that found with the reflex method. In fact Scobee[2] found that the deviation could disappear with the cover test when the child became nervous. In reality, then, the deviation was much greater for distance than for near in all cases even though the cover measurements were greater for near in most cases.

These findings indicate that cover measurements for distance and near alone cannot be relied upon for classification of intermittent exotropia.

### COMPARISON OF THE STRAIGHT-UP AND STRAIGHT-DOWN POSITIONS OF GAZE

As can be seen in Tables 4 and 5, the most consistent, and I believe the best, diagnostic findings in these cases is the comparison of the straight-up and straight-down positions. With the cover measurements, the exotropia looking up was greater in 93 percent of these cases than in downward gaze. Only in seven percent of cases were the measurements greater looking down.

Examination with versions was even more diagnostic since the exotropia found in sursumversion was greater than that in deorsumversion in 100 percent of the cases and the eyes were actually straight looking down in 70 percent.

These findings are especially important in young children who can fix on a light but cannot maintain fixation long enough to be measured accurately with the cover test.

### VERTICAL DEVIATIONS (table 4 and 5)

In 68 percent of our cases there was a bilateral vertical deviation found with cover measurements in which the right hypertropia was greatest on levo-elevation and the left hypertropia was greatest on dextro-elevation. In 13 percent of the cases only a unilateral vertical deviation could be measured but it was still diagnostic in that if a left hypertropia was present it was greatest in dextro-elevation and if a right hypertropia was present it was greatest in levo-elevation. Alternating sursumduction was frequently found in the primary position.

### LATERAL VERSIONS (table 5)

In 73 percent of the cases, bilateral elevation in adduction was observed. In 43 percent it was marked and grossly visible; in 30 percent it was moderate to minimal but easily recognized.

## Case reports

Two cases are presented to illustrate some of the problems of diagnosis and treatment.

### Case 1

L. B., aged three and one-half years, came to the Infirmary on April 12, 1948, with a history of the eyes turning out since birth. Her mother also had a marked exotropia since early childhood.

With atropine cycloplegia refraction was as follows: O.D., +0.25D. sph. ⊂ +1.25D. cyl. ax. 15° = 20/30; O.S., +0.5D. sph. ⊂ +1.25D. cyl. ax. 170° = 20/30.

Examination revealed 45 degrees of exotropia for distance and near (fig. 1A-c and f). Surgery was advised but the parents refused and left the clinic.

The child was returned to the clinic on August 15, 1950, two years later, for surgery. Examination revealed about 50 degrees of exotropia for distance and near (figure 1A-a and b). There was about 50 degrees of exotropia in sursumversion and about 25 degrees in deorsumversion. Bilateral elevation in adduction was well seen.

No near-point of convergence could be elicited when a light, pencil point, or small letter were used for fixation but, when minus 3.0D. lenses were placed in front of the eyes, a good near-point was found. Cover measurements were 66 prism diopters of exotropia for distance and 75 prisms diopters for near.

A diagnosis of exotropia with bilateral elevation in adduction was made and the child was admitted to the hospital for surgery.

Examination in the hospital on September 20, 1950, before surgery, revealed that the eyes could be straight for distance and near (fig. 1B). The near-point of convergence was 40 mm. There was 45 degrees of exotropia looking up while the eyes could be straight looking down. Bilateral elevation in adduction was present. The cover measurements were 50 prism diopters of exotropia for distance and 56 prism diopters of exotropia for near.

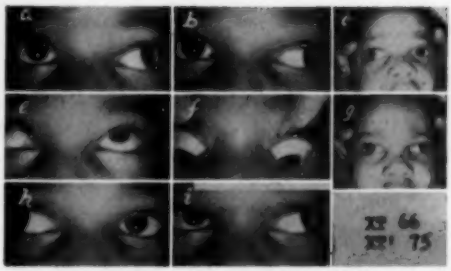



Fig. 1A (Urist). *Case 1.* Pre-operative photographs taken on April 12, 1948, and August 15, 1950.

a. 50 degrees left exotropia for distance.
b. 50 degrees left exotropia for near. No near-point of convergence could be elicited.
c. 50 degrees right exotropia for distance (photographed in 1948).
e. 50 degrees exotropia looking up.
f. 25 degrees exotropia looking down.
g. 50 degrees exotropia for near (photographed in 1948).
h. Eyes level on dextroversion (elevation in adduction seen well clinically).
i. Elevation of the right eye in adduction.

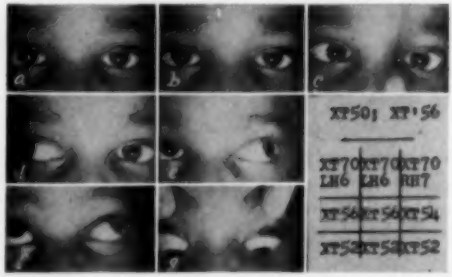



Fig. 1B (Urist). *Case 1.* Photographs taken on September 20, 1950, on examination just before surgery, when patient was in the hospital.

a. Eyes straight for distance. (Note large exotropia measurements.)
b. Eyes straight for near. (Note large exotropia measurements.)
c. Near-point of convergence 40 mm.
d. Eyes level on gaze to the right.
e. Elevation of the right eye in adduction on levoversion.
f. 45 degrees of exotropia on upward gaze.
g. Eyes straight looking down.

This case showed the variability of the squint as stressed by Scobee.[2] The photographs were all taken with the same lighting

set-up. Note that when the large exotropia was present the pupils were dilated (fig. 1A-a and b) and when the eyes were straight the pupils were small (fig. 1B-a and b). These variations in the squint and pupils indicated the marked innervational factor that was present.

The exotropia for distance was more than 50 degrees (fig. 1A-a) yet the cover measurements were only 66 prism diopters. This indicated that fusion and convergence were stimulated by the taking of the measurements.

The exotropia, the near-point of convergence, and the cover measurements were all very variable. The constant diagnostic findings upon which we placed great importance were the bilateral elevation in adduction and the greater exotropia looking straight up than straight down.

Fig. 2A (Urist). *Case 2.* Examination in November, 1947.

a. 25 degrees of exotropia and 10 degrees of hypertropia of the left eye for distance.

b. 25 degrees of exotropia and 10 degrees of hypertropia of the right eye for distance.

c. Eyes straight for near.

d. Slight elevation of the left eye in adduction.

e. Slight elevation of the right eye in adduction.

f. Near-point of convergence 40 mm.

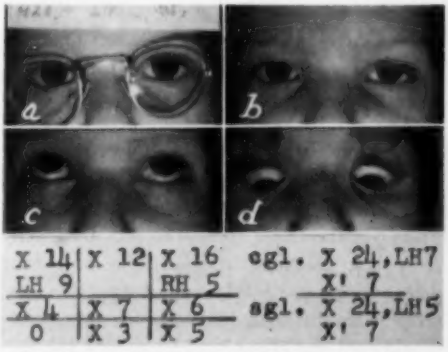



Fig. 2B (Urist). *Case 2.* Photographs taken on examination on August 15, 1952.

a. Eyes straight for distance and near with glasses.

b. Eyes straight for distance and near without glasses.

c. Eyes straight looking up.

d. Eyes straight looking down.

CASE 2

J. W., aged three and one-half years, came to the Infirmary in November, 1947, with a history of the left eye turning out for about six months.

With atropine cycloplegia, refraction was as follows: O.D., +0.5D. sph. ⌒ +1.25D. cyl. ax. 100° = 20/30; O.S., +0.25D. sph. ⌒ +1.0D. cyl. ax. 80° = 20/70.

Examination (fig. 2A) revealed that for distance, with the right eye fixing, there was about 25 degrees of exotropia and 15 degrees of left hypertropia, and with the left eye fixing there was 25 degrees of right exotropia and 15 degrees of right hypertropia. For near the eyes were straight. The near-point of convergence was 50 mm.

There was slight elevation of the left eye in adduction, the eyes were level on gaze to the left, and there was alternating sursumduction. Cover measurements were 25 prism diopters of exotropia with four prism diopters of left hypertropia for distance and nine prism diopters of exophoria for near. The patient had first-grade fusion.

Because of the exotropia, the atropine refraction was reduced by a −1.0D. sph. and the following correction was prescribed: O.D., −0.5D. sph. ⌒ +1.25D. cyl. ax. 100°; O.S., −0.75D. sph. ⌒ +1.0D. cyl. ax. 80°.

Following occlusion of the right eye, vision in the left eye improved to 20/30.

Examination on August 15, 1952, five years after the initial examination (fig. 2B), revealed that the eyes were straight in all positions. There was third-grade fusion at 0.

Cover measurements were those of exophoria only. The mother stated that, as far as she knew, the left eye had not been turning out for the past year or so. The child had no complaints and did her school work well.

When fusion was interrupted with the taking of cover measurements, a large exophoria was still present which was greatest on upward gaze and revealed the diagnostic vertical measurements (fig. 2B). The exophoria apparently caused no difficulty; the exotropia for distance had disappeared along with the elevation in adduction and the alternating sursumduction.

This case has been presented to show that these cases can improve with the wearing of glasses, the elimination of the amblyopia by occlusion, and the development of good fusion. It has not been my experience that all cases with an exotropia greater than 18 prism diopters for distance require surgery, as advocated by Scobee.[12] As long as good vision and fusion are maintained for near, there is no urgent need for surgery.

Note that in this case also the exotropia was 25 degrees and the hypertropia 15 degrees yet the cover measurements were only 25 prism diopters of exotropia and four prism diopters of left hypertropia for distance. This difference between the actual exotropia and the cover measurements for distance also seems to indicate that good fusion can be present. This was verified in this case in that although only first-grade fusion was found at the first examination, a later examination revealed the presence of stereopsis.

## Comment

Cases of exotropia with bilateral elevation in adduction comprised the majority of the cases of "intermittent exotropia" in my series. The squint usually started at birth, and in 80 percent, below the age of five years.

The refractive error appeared to have no statistical significance as to the etiology of the exotropia. The near-point of convergence was variable, but was less than 60 mm. in 55 percent of the cases. Cover measurements were greater for near than for distance in 60 percent which was contradictory, since these were "divergence excess" cases.

With the corneal reflex measurements, however, the exotropia was greater for distance than for near in 78 percent, and the same as near in 22 percent. In no instance was the exotropia greater for near than for distance by this method of examination. The explanation for this discrepancy between cover and reflex methods of examination could be that, with the taking of prism cover measurements, the child was stimulated to pay attention to the fixating light. This stimulated convergence and fusion which made the exotropia for distance measure less than it actually was when the child was gazing into the distance or day-dreaming.

Vertical deviations were found in 81 percent of the cases and were part of the clinical syndrome. Bilateral hypertropia was found in 68 percent of cases and unilateral hypertropia in 13 percent. Characteristically, the left hypertropia was greatest in dextro-elevation and the right hypertropia in levo-elevation. Bilateral elevation in adduction was seen in 73 percent of the cases. Alternating sursumduction was very commonly found in the primary position. Fusion and vision were usually well developed because of the intermittency of the squint.

In 88 cases in which the vision was recorded 82 cases had equal vision. Four cases had 20/70 in an amblyopic eye which was brought up to normal with a modicum of occlusion. One case had 20/200 and another had 5/200 with eccentric fixation in the amblyopic eye. In both of these cases the vision was brought up to 20/30 within six months by occlusion of the better eye.

## Summary

Most cases of "intermittent exotropia" can be classified into the group "exotropia with bilateral elevation in adduction." The

advantage of this classification is that it is based on added findings which proved to be more consistent and accurate than the diagnosis of divergence anomalies based only on the near-point of convergence and the difference between the prism cover measurements for distance and for near.

The important findings that make up the clinical syndrome are: (1) Bilateral elevation in adduction, (2) a greater exotropia for distance than for near, (3) a greater exotropia in sursumversion as compared with deorsumversion, and (4) characteristic vertical deviations which are, a left hypertropia that is greatest in dextro-elevation and a right hypertropia greatest in levo-elevation.


REFERENCES

1. Urist, M. J.: Horizontal squint with secondary vertical deviations. Arch. Ophth., **46**:245 (Sept.) 1951.
2. Scobee, R. G.: Intermittent exotropia. Tr. Am. Acad. Ophth. 1949, July-Aug., p. 658.
3. Bair, D. R.: Symposium intermittent exotropia: Diagnosis and incidence. Am. Orthoptic J., **2**:12, 1952.
4. Scobee, R. G.: The Oculorotary Muscles. St. Louis, Mosby, 1952, pp. 172-174.
5. Sugar, H. S.: Symposium intermittent exotropia: Treatment. Am. Orthoptic J., **2**:20, 1952.
6. Burian, H. M.: The principles of surgery of extraocular muscles. Am. J. Ophth., **33**:381, 1950.
7. Chavasse, B.: Worth's Squint. Philadelphia, Blakiston, 1939, p. 238.
8. White, J. W., and Brown, H. W.: Occurrence of vertical anomalies with convergent and divergent anomalies. Arch. Ophth., **21**:999 (June) 1939.
9. Costenbader, F.: Management of divergent strabismus: In Strabismus Ophthalmic Symposium. St. Louis, Mosby, 1952, p. 354.
10. White, J. W.: Discussion: Surgical treatment of heterophoria by Townes, C. D. Tr. Am. Acad. Ophth., 1945, July-Aug., p. 342.
11. Scobee, R. G.: The Oculorotary Muscles. St. Louis, Mosby, 1952, 168.
12. ———: Ibid., p. 172.
13. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, Mosby, 1949, v. 4, p. 4019.


## POSTNATAL CATARACTS IN PREMATURE INFANTS*

### REPORT OF TWO CASES


LOREN P. GUY, M.D.
*New York*


Little is known or has been written about acquired cataracts occurring without apparent cause in infants and children, in particular in those prematurely born. Trauma, uveitis, galactose disease, malnutrition, hypoparathyroidism, and exposure to X-ray or radium radiation are most prominent among the known causes of acquired cataracts. All of these are possible to identify.

At Bellevue Hospital, however, two prematurely born infants were observed to develop cataracts, between the third and sixth month after birth, without any apparent etiology. One child had pyloric stenosis and was quite ill but the second child was always in good health and no definite cause has been ascertained.

Since the condition may be rare or has not been recognized, yet is clinically important, the two cases are reported. I did the eye examinations and the diagnosis of cataracts has been confirmed by colleagues. The medical work on the patients has been done by Dr. Hans Kunz of the Children's Medical Service at Bellevue Hospital.


* From the Children's Medical Service, Bellevue Hospital, New York. Presented at the West Side Clinical Society, New York, on February 14, 1952, and at the New York Academy of Medicine on November 19, 1953. This study was aided by a grant from the National Society for the Prevention of Blindness.


NOTE: Since these two cases were reported, a third case has been found. The patient was prematurely born and was given much oxygen. The child has been healthy in all respects. This brings the incidence in our group to three in 1,000 cases.

CASE 1

C. M., male, Bellevue #23194-50 and #26282-50.

*Family history.* The mother was an 18-year-old Puerto Rican, Rh positive, Wassermann negative, in good health. The father, aged 21 years, Puerto Rican, was in good health. There is no history of ocular maladies in either of the parents or of their families.

*Prenatal and neonatal period.* This was the first pregnancy of this mother and was normal except for spontaneous termination after 34 weeks' gestation. Delivery on March 24, 1950, of a normal baby weighing 2,100 gm., or four lb. and 10 oz., was normal.

*Course in hospital.* The infant took feedings poorly from birth and appeared dehydrated on March 28, 1950, after vomiting and refusing the bottle. Several clyses of five-percent glucose in water and saline were given. Very mild diarrhea also developed on March 28, 1950. Chloromycetin was given on April 2 to 5, 1950, for possible septicemia which was never proven by blood culture. The weight dropped to 1,900 gm. or 4 lb. 3 oz. after birth but was being regained on March 28, 1950.

On April 10, 1950, he suddenly developed projectile vomiting and the next day the diagnosis of pyloric stenosis was made after palpation of an intra-abdominal swelling. He was given eumydrin (methyl atropine nitrate) but continued to vomit and to lose weight.

On April 18, 1950, he developed a flush, abdominal distention, and mydriasis due apparently to Eumydrin. On May 13, 1950, pyloroplasty was performed. There was an uneventful postoperative course and a cure of the pyloric stenosis. He was discharged from the hospital on May 21, 1950, in fair condition. A heart murmur was heard but was thought to be insignificant. No tetany was ever observed.

In addition to eumydrin and chloromycetin, vitamin K, oxygen, penicillin, monocillin, streptomycin, sodium phenobarbital, caffeine sodium benzoate, elixir of ferrous sulphate, and vi-penta vitamin drops were administered.

*Laboratory data.* Urine analysis was always normal except on May 25, 1950, there was an insignificant reaction (green color) to Benedict's test. Hemoglobin varied between 9.0 and 21 gm., the latter on May 14, 1950. Blood counts and stains were normal. Nose, throat, and urethral cultures showed no pathogens. Serum calcium was 9.2 mg. percent; phosphorus 4.96 mg. percent; and phosphatase 6.08 mg. percent. Blood sugar 83 mg. percent; tuberculin test negative; electrocardiogram normal.

*Eye history.* The ocular media and fundi were normal through dilated pupils on April 7, 1950, two weeks after birth. When the eyes were examined again on August 11, 1950, the lenses were found to be opaque. The greater opacity was central and nuclear but some was in the cortex. The child was referred to the ophthalmologic service and was admitted August 27th for cataract surgery and both eyes have been operated on. Esotropia and nystagmus have developed.

CASE 2

R. J. H., female, Bellevue #15115-51.

*Family history.* The mother was a 34-year-old, white woman, Rh positive, Wassermann negative, who had had two previous pregnancies with normal babies. The father, aged 36 years, white, was in good health. No history of eye disease in either parent or relatives.

*Prenatal and neonatal period.* Normal pregnancy and uncomplicated delivery on

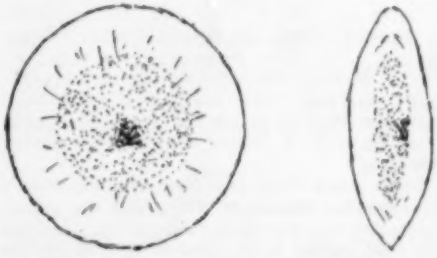


Fig. 1 (Guy). Schematic drawing of the cataracts found in two patients.

March 5, 1951, after 32 weeks' gestation. Birth weight 1,690 gm. or 3 lb. 9.5 oz.

*Course in hospital.* Perfectly normal. The infant was fed increasing amounts of Olac formula until March 23, 1951, when she was changed to evaporated milk. She gained weight satisfactorily and was sent home April 5, 1951.

Medication while in the hospital was oxygen, vitamin K, vitamin C (because of intolerance to orange juice), Drisdol vitamin drops, elixir of ferrous sulphate, penicillin, and aureomycin. The pupils were dilated three times with homatropine. No tetany was ever observed.

*Development.* She was smiling at one month, rolling over at three months, sitting at 11 months, and mentally bright.

*Laboratory data.* Hemoglobin 10.5 to 16 gm., blood count and blood sugar normal except once the white count was 12,900. Urine normal with no reduction of Benedict's solution. Fluoroscopy normal.

*Eye history.* On March 7, 14, and 28, 1951, the ocular media and fundi were normal through dilated pupils. On June 27, 1951, the child was not co-operative and the eyes were not satisfactorily examined so no opinion was given. On August 22, 1951, bilateral cataracts, predominantly nuclear with some cortical change, were identified. These have progressed slightly but at present seem stationary. The child has developed alternating estropia but no nystagmus. She is able to do most things other children do and so has not had cataract surgery.

## Discussion

These two children had two things in common, both were born prematurely and both had oxygen therapy. However, in the premature service all infants are premature and weigh less than five lb. or 2,500 gm. on admission and, at the time these two infants were in the hospital, practically all premature infants were given oxygen.

Except for the treatment for pyloric stenosis for the first infant, the treatments are those that are routine and standard for premature infants and were given to all of nearly 900 such patients whose eyes I have examined.

I have in my practice another child with similar cataracts, who was born prematurely but who had no eye examination until she was over a year old so that the question of whether her cataracts are congenital or postnatal cannot be answered.

Galactose cataracts that I have observed have been lamellar. When the disease was controlled the opacities decreased, some to normal appearance. The urine from galactosemia patients will reduce Benedict's solution and there are other signs of the disease such as enlarged livers and spleens, edema, and malaise. Neither of these two patients had galactosemia.

Tetany was not observed in either child and in the one child whose serum could be studied there was no evidence of calcium or of phosphorus dyscrasia.

One child (Case 1) was definitely malnourished due to pyloric stenosis but the other child was not. In several badly malnourished children whose eyes I have studied there have been no cataracts. In one group with deprivation of riboflavin none of the patients had cataracts. Neither child was subjected to ocular trauma nor to radiation.

The first patient had massive doses of methyl atropine nitrate which at the time was a drug that was new to me. Frankly, I suspected it as being a probable cause of the cataracts, recalling the numerous cataracts that were caused by naphthalene when that was a popular drug for overcoming obesity. However, the second patient took no methyl atropine nitrate and thus disproved this idea. Furthermore, other patients have taken the drug without developing cataracts.

Colleagues who examined these children called the cataracts congenital until the histories were made known. It may be possible there have been more cases and they may have been considered congenital.

My attention to the eyes of premature children has come about in trying to learn something of retrolental fibroplasia. This disease has caused the eyes of infants, especially premature infants, to be studied earlier and more thoroughly than formerly. Finding these two cases is a by-product of that study.

In the past it has been the practice to have premature infants handled as little as possible so their eyes have not been studied as extensively as in the past five or 10 years so similar cataracts may have long occurred but have not been recognized as being postnatal in development.

I have not studied the eyes in a comparable series of full-term babies and so I have no basis for an opinion as to whether these postnatal cataracts are limited to premature infants.

If this is a common disorder and we have wrongly classified some cataracts as being congenital, then our ideas on the heredity factors of these cataracts will have to be revised. Advice about marriage and having children will have to be different from what it has been.

As to treatment, one argument for the early operation of congenital cataracts is the possibility of stimulating the macula to function. These two children saw before they developed cataracts so their maculas have possibly been urged to function. The child who has not had surgery is as well off as the one who has and runs less risk of developing retinal separation and other complications.

### Conclusions

1. Postnatal cataracts have been observed to develop in two prematurely born infants.
2. As yet no cause for these cataracts has been established.

*40 East 62nd Street (21).*

---

## AN APPARATUS FOR INVESTIGATION OF THE EYE*

### In monochromatic light with interference filters

M. Hultin, Ph.D., and T. Kornerup, M.D.
*Stockholm, Sweden*

In ophthalmoscopy the light employed is mainly "white" light, that is, light in which all qualities of the spectrum are represented with about the same intensity. The advantages of light-sources, in which the spectral distribution is altered in one way or another became apparent quite early.

Such "colored" light may be produced in in three fundamentally different ways, in each case giving a light of a certain color and with a varying degree of purity.

1. Bezold and Engelhart (1877) used the short-wave part of a prismatic spectrum for special purposes in which connection they observed a sharp contrast between the retinal vessels and the fundus of the eye. The same method was later used by Helmbold (1910), Ginestous (1911), and von Heine (1918).

2. The second method implied the use of filters (Valentin, 1878; Neuschüler, 1897, 1898). Since Vogt described his method of examining the fundus with carbon-arc light deprived of its red components by passage through an erioviridin copper-sulfate filter, this red-free light has assumed great importance in investigations of the fundus. This method or its modifications have been used by Dobson (1928, 1929), Friedenwald (1924, 1927, 1928), von Heine (1918), Lau-

* From the Institute of Physics, University of Stockholm, and the Ophthalmic Clinic, Caroline Hospital.

ber (1922, 1929), and Müller and Franceschetti (1933).

3. The third method involves light-sources with discontinuous spectra, for example, light due to lithium, calcium, and barium (Valentin, 1878), sodium (Tschernning, 1898; Serr, 1937; Pavia, 1941) or mercury (Mayou, 1903; Gullstrand, 1906).

By using colored light in ophthalmoscopy all these authors achieved purely qualitative results. The arterioles were more distinctly seen; minute, hardly visible vessels became visible; pigment and hemorrhages could be distinguished from each other. Concerning the color of the macula, many interesting observations were made.

These results are due to the fact that different structures stand out with varying degrees of distinctness when they are seen in light of different wave-lengths. Such distinctness depends upon their properties of absorption and reflection. The different aspects under which certain structures appear in variously colored light have raised the question as to the wavelength at which a change occurs and the manner in which such changes take place.

Kugelberg (1937), the first to attack this problem, chose as his light-source a prism-monochromator, permitting of continuous variation of the wavelength. With this apparatus he undertook experiments to ascertain whether a detail in the fundus of the eye changed when observed in light of different wavelengths, and he also determined the wavelengths at which such changes occurred.

Thus, when observed in green light a retinal vessel appeared bluish dark on a green background. When the wavelength of the light was changed toward the red part of the spectrum, the vessel reflected an increasing amount of light. At the moment when the vessel and the background reflected the same amount of light the vessel seemed to disappear. At this point the wavelength was read on the monochromator. This wavelength was called the L-value.

When the prism of the monochromator was turned in the opposite direction, a certain position was reached at which the vessel again became visible. This new wavelength was called the K-value. The mean of these two values was called the LK-value and this value was determined by Kugelberg for many details of the ocular fundus in normal subjects.

In 1947, Kornerup examined the LK-values for the vessels of the human eye in diseased conditions. This study covered both retinal vessels and the external vessels of the eye. Special attention was devoted to the last-mentioned vessels, which form the pericorneal or ciliary injection in iritis, scleritis, keratitis, secondary iritis following penetrating wounds. The LK-values for this network of vessels were determined every second or third day throughout the course of the disease, and the behavior of the LK-value was compared with the clinical course.

It was found with almost complete certainty that a rise in the LK-values meant a deterioration of the clinical state and vice versa. In several cases, a rise in the LK-value preceded a deterioration. Hence, the determination of the LK-value proved to be an objective method of evaluating the course of the disease.

This was found to be of the greatest importance in connection with penetrating wounds when enucleation was considered. A series of steadily rising LK-values or a series of constantly high values indicated enucleation; whereas, a series of decreasing values was regarded as grounds for postponement of the enucleation. The variations in the LK-values were shown to depend upon variations in the oxygen-saturation of the vascular blood.

In the same paper (Kornerup, 1947), the LK-values of the retinal vessels were determined. It was found that this value was definitely higher in venous than in arteriolar determinations. The LK-value thus seemed to be a means of distinguishing fine arteriolar branches from venous ones. Simi-

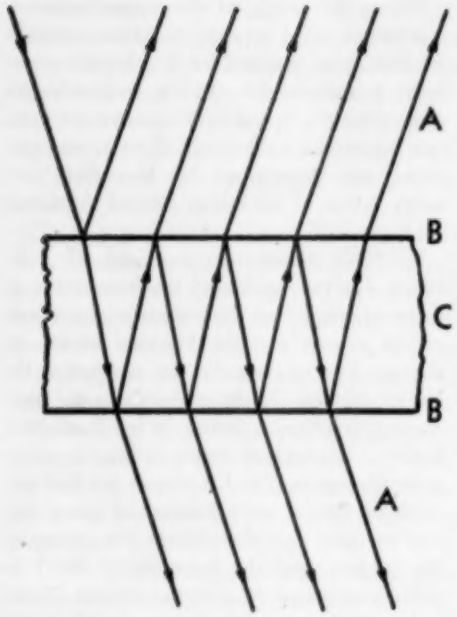



Fig. 1 (Hultin and Kornerup). Multireflection in the interference filter. (A) Glass cover. (B) Metal plate. (C) Dielectric medium.

larly, the determination of the LK-value yielded interesting results as regards the state of the walls of the retinal vessels in arteriosclerosis, hypertension, and diabetes.

The apparatus used by Kugelberg (1937) and by Kornerup (1947) was, however, rather inconvenient, and the introduction of this unwieldly instrument into general clinical practice was quite out of the question. A smaller and more convenient apparatus was needed; one that might be included in the equipment of the consulting room.

This seemed possible by using interference filters. These filters have three properties of value for the present problem:

1. The intensity of the transmitted light is comparatively high.

2. The monochromasy of the light is very good.

3. The wavelength of the transmitted light may to a certain extent be shortened by tilting the filter in such a way that the incident light strikes the surface of the filter at an oblique angle.

The principle of these filters is that of the interferometer of Fabry-Perot. In this instrument two parallel semitransparent metal plates are separated from each other by a relatively thick layer of air. The instrument is used for measuring distances and works with monochromatic light. On account of the relatively large distance between the plates high orders of interference are used.

The interference filters, on the other hand, are mainly intended for white light and work with lower orders of interference. The distance between the metal plates must therefore be small. This entails a considerable difficulty, namely, that of producing extremely thin dielectric media instead of air between the plates.

This practical problem has only recently been solved. Some transparent dielectric media with a low index of refraction, for example, fluoride of magnesium, are included between the two metal plates, and these in turn are covered by protecting glass plates. (Distance between the plates 0.0003 mm.)

When a bundle of light strikes the surface of the filter, some part of the incident light will be reflected at the first metal plate, while another part will penetrate the dielectric medium and strike the other metal plate, whereupon there is a similar splitting up of the light, some passing through the thin metal plate and another part being reflected back into the dielectric medium, and so on. Figure 1 gives an idea of this repeated reflection.

If the incident light strikes the filter at right angles, both the penetrating and the secondarily reflected light will coincide. The different components have, however, passed unequal distances through the dielectric medium an unequal number of times, and the waves are consequently displaced as regards the phase. The result will be an interference extinguishing certain wave lengths, while others will pass the filter with a negligible loss of intensity.

The intensity of the emergent light, I,

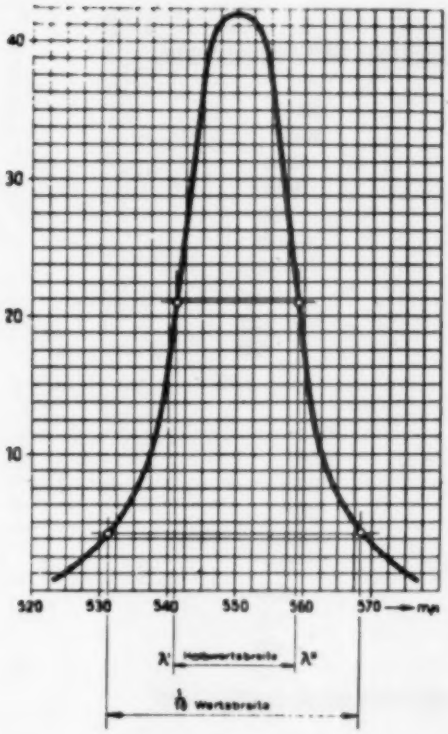



Fig. 2 (Hultin and Kornerup). Transmission curve of interference filter 550 mμ. The wavelength range λ'-λ'' marks the half-band width of the filter. (From "Interference filters" by Gerätebau-Anstalt Balzers, Liechtenstein.)

will vary according to the transmission, T, and the reflecting power, R, of the metal plates, and finally to the difference in phase ϑ.

$\vartheta = 2\pi\lambda_0/\lambda$ and $\lambda_0 = 2h_0 n$ where $h_0$ is the thickness and n the index of refraction of the dielectric medium. Then

$$I = \frac{T^2}{1+R^2-2R\cos Y_0\delta}$$

where $Y_0$ = intensity of incident light

If I is determined for different values of λ a characteristically steep intensity-curve will be obtained (fig. 2). This curve with its narrow half-band width, which is a good expression for the spectral purity of the transmitted light, is well seen in a spectrophotogram recorded with the apparatus described by Kornerup (1952). The apparatus was here modified for absorption-spectrograms. The formula just given shows the influence of the reflecting and transmitting powers of the metal layers on the steepness of the curve.

As may be seen, the angle of incidence plays an important role for the phase. If the light strikes the filter at an oblique angle, the maximum of transmission is dislocated toward shorter wavelengths. If the angle of incidence is α, the wavelength of the maximum λ changes as follows:

$$\lambda = \lambda_0 \sqrt{1-\frac{\sin^2\alpha}{n^2}}$$

where $\lambda_0$ = the maximum of transmission at an angle of incidence = 0°, this wavelength being used as a denomination of the filter.

The tilting of the filter is, however, followed not only by a shortening of the wavelength. At the same time the intensity of the transmitted light is reduced and the monochromasy is to a certain extent vitiated.

The series of filters used for the following ophthalmologic work consists of eight filters. The wavelength number for the maximum transmission serves as a denomination for the filter in question. Thus a filter 636 has its maximum transmission at 6,360 Å when the incident light strikes the filter at right angles. When tilted 30 degrees, the

Fig. 3 (Hultin and Kornerup). A spectrogram of an interference filter recorded with phototube GP 22.

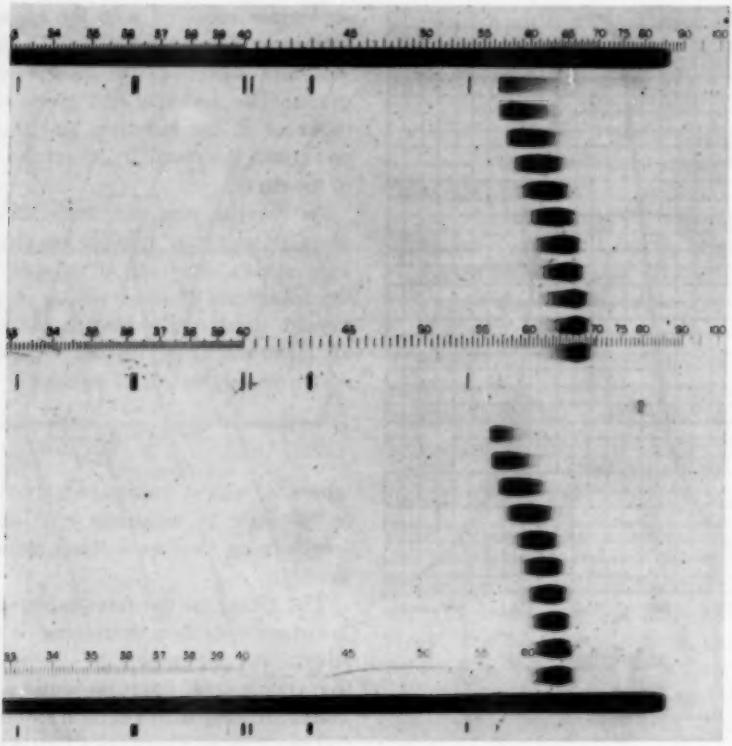


Fig. 4 (Hultin and Kornerup). Spectrograms recorded with a medium quartz spectrograph for the filters 636 and 669.

emergent light has a wavelength of 5,930 Å, and when tilted 40° the emergent light has a wavelength of 5,640 Å.

The filters should not be tilted more than 30 degrees. When this degree is passed, the intensity of the emergent light is diminished and the filter is no longer monochromatic. Finally, interferences of higher degrees will appear. In short, the accuracy of the filter is lost. This is well seen in Figures 4 and 5.

Figure 4 shows spectrograms recorded with a medium quartz spectrograph for the filters—636 and 669. As light-source a wolfram-band lamp of 8.5 volts was used. The filters were placed between the condensing lens and the entrance slit. The photographic plate used was Ilford long range, sensitive up to 8,500 Å. Exposure 10 seconds.

For purposes of comparison a mercury spectrum and ordinary white light are exposed on the same plate. The spectrograms are taken at every fifth degree from 0 to 50 degrees. These show the displacement of the maximum of transmission, the broadening of the transmitted band as well as the lowering of the intensity, and, at 40 degrees, the appearance of interferences of higher orders.

A spectrophotometric analysis on a Beckman photometer of interference filters gives curves of the kind shown in Figure 5, obtained with a filter 580.

Figure 6 shows the wavelength range covered by the series of filters used, namely 4,770 to 6,860 Å. The curves are obtained by wavelength measurements on spectrograms of the kind seen in Figure 4. Thus, filter 512 covers the range from 4,770 Å

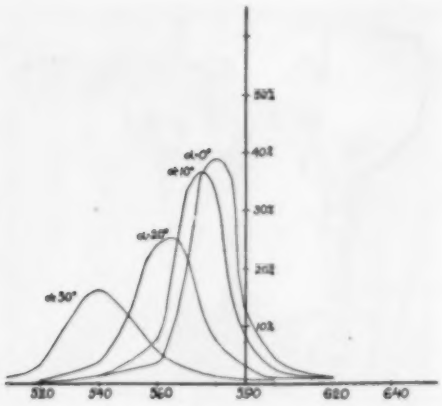


Fig. 5 (Hultin and Kornerup). The curves are registered with a Beckman photometer. With increasing angle the maximum is displaced toward the short-wave part of the spectrum. The intensity and spectral purity are, at the same time, reduced.

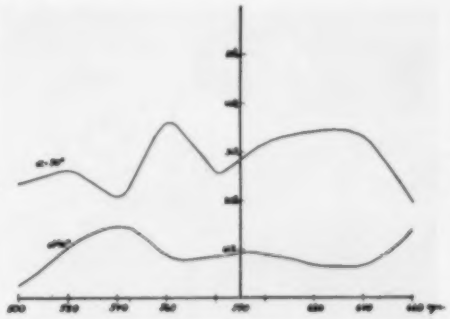


Fig. 5a (Hultin and Kornerup). When the angle of incidence is greater than 30 degrees, the regular shape of the curves is lost and the filter is no longer reliable for practical purposes.

up to 5,120 Å, filter 547 covers the range from 5,090 Å, up to 5,470 Å, and so on.

The filters are seen to overlap, and one and the same wavelength may be obtained with two or three different filters. For instance, wavelength 6,160 Å is obtained with filter 616 at 0 degrees, but also with filter 636 at 20.6 degrees, and again with filter 658 at 29.4 degrees. However, as already mentioned, the half-band width and the intensity of the transmitted light are quite different in the three cases.

For routine ophthalmologic work with interference filters a special lamp has been built.*

The lamp (fig. 7) consists of a light-source, an eight-volt spiral lamp of 48 watts. A condensing lens 28D. placed 3.5 cm. from the lamp throws parallel light toward the filter. This is held in a frame

* We are very indebted to Mr. Evert Aulin at the Aga, Ltd., in Stockholm for his kind advice and assistance. The first lamp was built at the Aga Factories, Lidingö, Sweden.

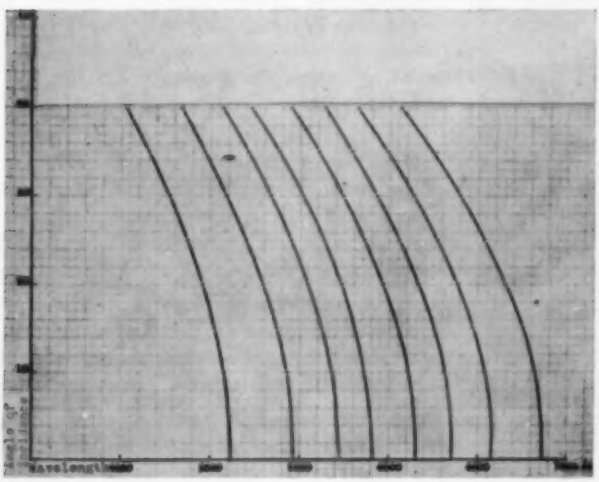


Fig. 6 (Hultin and Kornerup). The relation between the angle of incidence and the wavelength for interference filters. The curves are derived from spectrograms of the type shown in Figure 4.

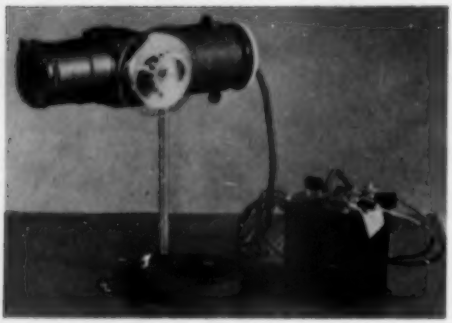


Fig. 7 (Hultin and Kornerup). View of the lamp.

with metal springs, and the frame may be tilted by means of a cogwheel system. The tilting angle is read on a transparent scale illuminated from behind. In front of the filtercase there is a variable diaphram. The whole apparatus is very handy and small; being 23 cm. in length and 26 cm. in height.

In ophthalmologic work it may be used as:

1. A light-source for direct ophthalmoscopy with the Morton reflex ophthalmoscope.

2. A light-source in biomicroscopy of the eye, when the lamp is directed toward the eye and the ciliary vessels are observed by means of a loupe or a binocular low-power microscope.

3. A light-source for the reflection-free binocular ophthalmoscope of Gullstrand (fig. 8).

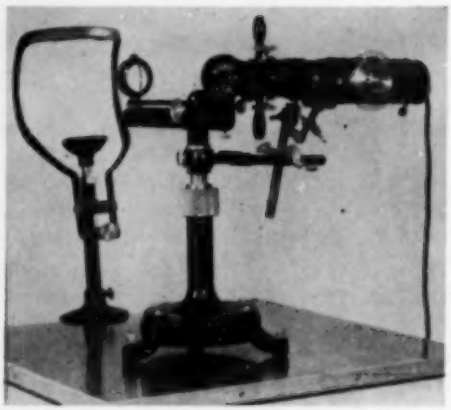


Fig. 8 (Hultin and Kornerup). Light source for the reflection-free binocular ophthalmoscope of Gullstrand.

The lamp may also be used for several photographic purposes and in chemico-analytical work. It has also proved to be useful in microscopy, but practical experience of microscopy in monochromatic interfering light is, so far, limited.

The interference lamp is consequently not only a substitute for the ordinary prism monochromator; it is smaller and handier than the latter and gives higher intensity without any loss in spectral purity. It is also much cheaper.

*Ophthalmic Clinic*
*Caroline Hospital.*

## References

Bezold and Engelhart: Ber. d. könig. bayr. Acad. d. Wiss., **7**:226, 1877.
Dobson: Am. J. Ophth., **11**:431, 1928.
———: XIII. Concil. Ophth. Holland, **1**:36, 1929.
Friedenwald, J. S.: Am. J. Ophth., **7**:940, 1924.
———: Bull. Johns Hopkins Hosp., **40**:201, 1927.
———: Tr. Am. Ophth. Soc., **26**:381, 1928.
Ginestous: Rec. d'opht., **33**:248, 1911.
Gullstrand: Arch. f. Ophth., **62**:1, 1906.
v. Heine: Arch. f. Ophth., **97**:271, 1918.
Helmbold: Med. Klin., **6**:1649(2) 1910.
Hultin: Sveriges univ. o. högskole. am. förb. tidskrift, **2**:25-30, 1952.
Kornerup: Acta Ophth., **24**:423, 1946.
———: An investigation, in successively variable monochromatic light, of vessels of the human eye in diseased conditions. Acta Ophth., Suppl. XXVIII, 1947.
———: Svenska läk.-sällsk. Handl., **72-73**:39, 1950-51.
Kugelberg: Ophthalmoskopische Studien in monokromatischem sukzessiv veränderlichem Licht. Diss. Uppsala, 1937.

Lauber: Klin. Monatsbl. f. Augenh., **68**:226, 1922.
———: XIII. Concil. Ophth. Holl., **1**:36, 1929.
Mayou: Tr. Ophth. Soc. U. Kingdom, **23**:390, 1903.
Müller and Franceschetti: Klin. Monatsbl. f. Augenh., **90**:633, 1933.
Neuschüler: Rec. d'opht., 1897, p. 643.
———: Rec. d'opht., 1898, p. 564.
———: Bull. Soc. franç. d'opht., **16**:499, 1898.
Pavia: Fondo de ojo a la luz de sodio. Buenos Aires, 1941.
Serr: Arch. f. Ophth., **137**:636, 1937.
Tscherning: Bull. Soc. franç. d'opht., **16**:500, 1898.
Valentin: Moleschotts Untersuch. Ztschr. Natur-Lehre, **12**:31, 1878.

---

# LOSS OF THE LOWER FORNIX OF THE ANOPHTHALMIC SOCKET

ALSTON CALLAHAN, M.D.
*Birmingham, Alabama*

The most frequently encountered deformity of the socket is the loss of the lower fornix from contracture, and it is often accompanied by relaxation ectropion of the lower lid (fig. 1). The constant chief complaint is inability to retain a prosthetic eye.

This condition may result from failure to insert or wear a conformer soon after the eye has been removed, or it may develop at any time—early or late—after the eye has been removed because of the failure of the patient to keep a prosthetic eye in the socket. Wearing a prosthetic eye which is too large will cause this condition. It has also been reported as developing in some sockets even when the prosthetic eye apparently was normal in size. It may follow destruction of the eye and conjunctiva from severe injuries, such as molten-metal burns of the lower fornix.

In the *Ophthalmic Review* of 1893, Maxwell[1] described this defect and mentioned its frequency. To correct it, he used a hammock-like flap of skin from the anterior surface of the base of the lower lid, detached except at the ends, inverted, and sutured so as to form a new lower fornix. He found this method preferable to that, which he had used previously, which employed a free mucosal graft from the esophagus of a cat. Ten years later, he[2] reported this skin-inversion method again. However, in neither of his articles did he discuss the deformity of the lower lid which it causes, nor did he indicate the undesirability of having a socket lined partly with skin and partly by mucosa.

Many methods of solving this problem have evolved since then. They include many types of sutures, mucosal grafts, and silver and rubber fixation plates. Portman[3] used mucosal grafts to restore the lower fornix in an anophthalmic socket. Henderson[4] used mattress sutures to draw the conjunctiva down to form a new lower fornix, the sutures being brought out through the skin below the eyelid.

Greeves[5] dissected a pocket down to the

Fig. 1 (Callahan). Ectropion and loss of lower fornix in anophthalmic socket.

inferior orbital rim through a horizontal conjunctival incision 10 mm. below the lower lid margin. The three mattress sutures used to close the incision were inserted through the periosteum of the rim and tied on the skin surface over a strip of rubber. This procedure was also later reported by Berens[6] and by DeVoe.[7]

Sherman[8] has combined with the insertion of a secondary implant the use of mattress sutures over a rubber strip to pull down the mucosa to the inferior orbital rim and this combination of the two is the ideal procedure if removal of the eye without the insertion of an implant has been done.

In many of my patients with this condition, however, a spherical implant is present. The technique which I have evolved and have now used successfully in 13 cases in the past five years is as follows:

An incision is made through the palpebral conjunctiva at the base of the lid. The exact site varies, being placed at the inferior edge of the tarsus if the lid is relaxed and ectropion is present. Postoperatively, cicatricial tissue will then extend from this incision to the inferior orbital rim and assist in correcting the ectropion. If ectropion is not present, the incision is made five to six mm. lower, at the site where the inferior fornix should be.

Whichever level is used, the incision is then extended parallel to the lid margin to within a few millimeters of the canthal angles. The thick and extensive cicatricial tissue often present along the floor of the orbit is entirely removed. The dissection is continued inferiorly and anteriorly with blunt scissors so as to expose the periosteum of the inferior orbital rim. Unless caution is exercised to keep the handles of the scissors depressed at an angle so that the blades are angled anteriorly toward the orbital rim, the dissection may proceed toward the orbital apex.

Even if the socket is not markedly contracted, I prefer to insert a mucous membrane graft for the reconstruction of the fornix than to attempt to shift the mucosa from the fundus downward. An elliptical section of buccal mucosa, about 25-mm. long and 15-mm. wide at its midpoint, is obtained from the inside of the lower lip with the lip clamp. It is unnecessary to close the donor defect tightly, but the edges should be brought toward each other with sutures.

The sector of oral mucosa is placed horizontally in the dissected fornix and sutured with interrupted sutures (4-0 silk or chromic gut).

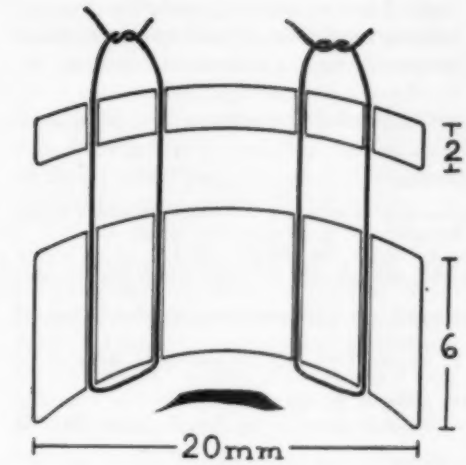
Two specially designed acrylic bars are now used (fig. 2). The bar for the outside is 2.0 mm. by 6.0 mm. by 20 mm., is slightly curved on the flat side, and has two pairs of perforations through the two-mm. thickness. Its purpose is to avoid erosion of the skin where the sutures are tied. The bar for the inside has the same measurements, but it is curved on the edge and its perforations are placed through the six-mm. thickness. Its function is to keep the mucosal graft firmly in contact with the surrounding structures and to assure its nutritional union to them.

A double-armed silk suture (4-0) is threaded through each of the two pairs of holes in the inside bar and with medium sized half-curved sharp-pointed needles, such as the Ferguson No. 514, No. 6, the sutures are carried through the skin (lowest drawing in Figure 2).

Both pairs of sutures are brought through the openings in the outside bar and drawn up and tied. This is done with sufficient tension to hold the graft firmly against the periosteum but not enough to risk the danger of sloughing of the lid (fig. 3).

The sutures uniting the graft and recipient conjunctival edges are removed after seven days if silk was used. The mattress sutures and bars are removed after 14 days. The inferior fornix is re-established and if ectropion has been present, this procedure should have corrected it (fig. 4).

A vertically elongated conformer designed



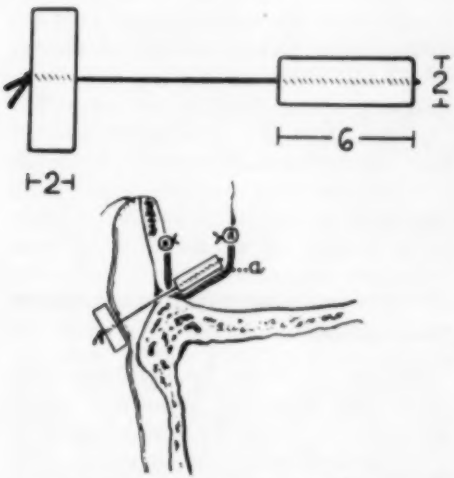




Fig. 2 (Callahan). Diagram shows construction of acrylic bars and the manner of suture insertion. The lower diagram indicates the manner that the bar presses the graft to the periosteum of the inferior orbital rim.

Fig. 3 (Callahan). Reconstruction of lower fornix and correction of ectropion. Mucous membrane graft was held by acrylic bar placed inside of the lid. It is fixed in position to the acrylic bar on the outside with sutures.

Fig. 4 (Callahan). Appearance after two months showing lower fornix and correction of ectropion. Cicatricial tissue holds the graft to the inferior orbital rim.

Fig. 5 (Callahan). Prosthetic eye remains in easily. It should be kept in the socket at all times.

for use in the correction of this deformity is worn after the removal of the bars to maintain pressure of the mucosal graft toward the inferior orbital rim. This is worn for three or four weeks until the newly formed lower fornix has united well to the inferior orbital rim. A well-fitting prosthetic eye should then be worn at all times (fig. 5).

*903 South 21st Street.*

## References

1. Maxwell, P. W.: Operation for relief of symblepharon or to enlarge a contracted eye socket so that it will hold a glass eye. Ophth. Rev., **12**:189, 1893.
2. ———: An operation to enlarge a contracted socket, etc. Ophth. Rev., **22**:121, 1903.
3. Portman, A. E.: Late implant of a glass ball in orbit, and epithelial lip grafts transplanted to orbit. J.A.M.A., **37**:978, 1901.
4. Henderson, T.: Suture operation for contracted socket and allied conditions. Tr. Ophth. Soc. U. Kingdom, **38**:119, 1918.
5. Greeves, R. A.: In discussion of paper by R. R. Cruise, "Operation for contracted sockets." Tr. Ophth. Soc. U. Kingdom, **39**:118, 1919.
6. Berens, C.: Restoration of the lower cul-de-sac. Am. J. Ophth., **26**:119, 1943.
7. DeVoe, A. G.: Experiences with the surgery of the anophthalmic orbit. Am. J. Ophth., **28**:1346, 1945.
8. Sherman, A. E.: The retracted eye socket. Tr. Am. Ophth. Soc., **48**:615-635, 1950.

---

# RETROBULBAR INJECTION OF LIDOCAINE (XYLOCAINE®) FOR ANESTHESIA AND AKINESIA*

DAN A. RUSSELL, JR., M.D.
*San Antonio, Texas*

AND

JACK S. GUYTON, M.D.
*Detroit, Michigan*

Observations on more than 200 cases in which Xylocaine,® a relatively new local anesthetic agent, has been injected for various ophthalmic operations, and on a number of eyes where Xylocaine has been instilled for topical anesthesia, have convinced us that the desirable or superior qualities claimed for this drug are generally valid. This report is concerned primarily with comparing the effectiveness of retrobulbar injections of Xylocaine solutions with that of procaine solutions.

* From the Wilmer Ophthalmological Institute of The Johns Hopkins University and Hospital. Presented at the Wilmer Residents Meeting, The Johns Hopkins Hospital, March, 1953.

® Xylocaine is trademark of Astra Pharmaceuticals, Inc., for their brand of Lidocaine.

## Retrobulbar anesthesia and akinesia

### Background

The use of cocaine for instillation anesthesia was introduced to medical science in September, 1884, by Koller,[1] an Austrian ophthalmologist. In December, 1884, H. Knapp[2] reported injecting 0.4 cc. of four-percent cocaine (16 mg.) behind the eye as a preliminary to enucleation, which was then carried out with complete freedom from pain.

If more than 20 mg. of cocaine is injected into an individual who is unusually susceptible to its effects, there is danger of obtaining a severe, even fatal, toxic reaction.[3] When cocaine was injected in the larger quantities required for general surgical pro-

cedures, a number of deaths occurred. These fatalities had an inordinate dampening effect on the ardor for injecting cocaine, even in the small dosage required for nerve-block anesthesia of a small structure such as the eye. Indeed, fear of injecting cocaine, even in dosage below the minimal lethal dose ever recorded, persists to this day.

By the time procaine was introduced (1905), ophthalmologists largely had come to rely alone on topical applications of cocaine for local anesthesia of the eye. This is hardly surprising, since repeated instillations of four-percent cocaine solution into a non-inflamed eye gives rather good anesthesia even of the iris. Also, the usual technique of cataract extraction, commonest of the intraocular operations, then consisted of combined extracapsular extraction without sutures—a technique which is considerably less time consuming and less demanding on extraocular muscle akinesia for prevention of vitreous loss than is now the case.

Procaine has so high a ratio of anesthetic to toxic dosage as to almost preclude any danger of a systemic reaction from the small amount it is feasible to inject retrobulbarly, especially if adrenalin (which slows absorption) is included in the injection. True, rare idiosyncrasies have been reported where fatality resulted from as little as 10 to 130 mg. of procaine, but such deaths have been immediate affairs, with a sudden collapse quite different from the toxic symptomatology of overdosage.[3] In general, it is quite safe to inject 1,000 mg. or more of procaine into the tissues if the solution is not too concentrated and contains some adrenalin; and the usual amount injected retrobulbarly is well below even the toxic dosage for intravascular injection.

At first, however, procaine injections for ophthalmic surgery were largely confined to operations on the ocular adnexa. Later, subconjunctival procaine injections began to be used for intraocular surgery as a supplement to the sensory anesthesia obtained from cocaine instillations. Still later, use of procaine to produce partial or complete akinesia of the orbicularis, either from injections within the muscle (Van Lint, 1914) or alongside branches of the seventh nerve (O'Brien, 1927), gradually became a widespread adjunct to cataract extraction.

The retrobulbar injection of procaine for intraocular surgery appears to have been used by relatively few men until the early 1930s. Probably more complicated surgical techniques, as well as widespread substitution of tetracaine in place of cocaine for instillation anesthesia (which resulted in decreasing the average depth of anesthesia of the conjunctiva and iris), thereafter began to force many ophthalmologists to rely more and more on retrobulbar procaine injections for adequate sensory anesthesia.

Still, the importance of complete akinesia of the extraocular muscles during intraocular surgery was generally overlooked. This was due at least in part to a reluctance of many surgeons to part with a time-honored belief that a "soft eye" renders it more difficult to make a section or to deliver a cataract. Therefore, a sufficiently concentrated solution of procaine to obtain the maximum motor nerve block from the small volume feasible for injection into the limited available space in the orbit, even though any possible systemic reaction is almost precluded by this limitation, was not used. Instead, less concentrated solutions, which had proven suitable for sensory nerve block in general surgery (usually procaine, one or two percent, containing adrenalin, 1 : 50,000 or less) were and very generally still are injected retrobulbarly. Actually, it is more difficult to block a motor nerve than a sensory one, and the smaller sensory fibers of pain and temperature are blocked by lower anesthetic concentration than are the larger ones of touch and proprioception.[3]

Some nine years ago, we began injecting procaine retrobulbarly in varying concentrations and amounts. From trial and error, we finally concluded that 1.5 to 2.0 cc. of a four-percent procaine solution, containing

adrenalin in a concentration of 1:20,000, represented the average optimal injection for relaxing extraocular muscles without unduly increasing the intraorbital fluid volume. Since that date, this dosage has been used more or less routinely at the Wilmer Ophthalmological Institute, even though it obviously fell far short of the ideal for retrobulbar injection.

There is a generally held belief among ophthalmologists that adrenalin should be omitted from the retrobulbar injection in the face of hypertensive or other cardiovascular disease. However, general medical opinion indicates the small amount of adrenalin in such an injection serves as more of a safeguard than a danger, and that no complications other than momentary tachycardia should be expected. Therefore, for some years adrenalin 1:20,000 has been used, even in patients with hypertension or other cardiovascular disease, especially since it increases the length of anesthesia and tends to minimize any orbital edema.

The ideal anesthetic solution for retrobulbar injection should possess the following characteristics:

1. It should spread rapidly and thoroughly through the orbital tissues, and should temporarily paralyze conduction through all the intraorbital nerves, motor as well as sensory, even if the injection is not made exactly within the muscle cone.

2. The temporarily complete nerve block should last several hours, or at least longer than any surgery undertaken (with allowances for complications). It goes without saying there must also be complete return of all nerve conduction afterward.

3. The injection should not cause appreciable increase in the water content of the orbit, because that would counterbalance the desirable effects of extraocular muscle akinesia in reducing pressure on the globe. Inasmuch as an average of 1.0 mm. proptosis results from the injection of each 0.75 cc. normal saline retrobulbarly, no more than 2.0 cc. of solution should be required for a maximal effect. Furthermore, the solution should not be locally irritating to tissue, such as to cause edema or vasodilation within the orbit.

4. The amount required for retrobulbar injection should be so far below the minimal lethal dosage as to preclude the possibility of a systemic toxic reaction, even if an injection inadvertently be made into a blood vessel.

In attempting to find a better drug for injection, we tried 1.0 cc. of two-percent tetracaine (containing adrenalin 1:20,000) in a considerable number of cases, and 1.5 cc. of two-percent Monocaine (containing adrenalin 1:20,000) in another series. In comparing these drugs with procaine, neither appeared to have advantages which were not outweighed by their disadvantages.

When "the spreading factor," hyaluronidase, was first reported (1931), the use of it with local anesthetics was immediately suggested. This was tried by Atkinson,[4] in 1936, on animals but, because of tissue irritation from impurities in the drug, he abandoned its use at that time. In more recent years, hyaluronidase has been greatly purified and rendered much less irritating to tissue, and is now used extensively with local anesthetics.

In 1949, Atkinson[4] reported on the use of hyaluronidase in a solution containing procaine (two percent) and adrenalin (1:10,000) for retrobulbar injection in 107 cases. He concluded the akinesia of the extraocular muscles was better, and the duration of the effect was about the same, as with injection of a similar procaine-adrenalin solution not containing hyaluronidase. However, in a recent personal communication, Dr. Atkinson says he injected larger amounts of the solution containing hyaluronidase, utilizing pressure on the lids to reduce orbital fluid content, and suggests this may have accounted for the equivalent duration of effects. Lebensohn[5] and Tassman[6] have also reported akinesia of the extraocular muscles is more complete when hyaluronidase is used.

Following Atkinson's report, we began adding Wydase, a purified brand of hyaluronidase, 7.5 u./cc. to the solution containing procaine (four percent) and adrenalin (1:20,000) which we were accustomed to injecting. At first, we were enthusiastic, but we gradually gathered the clinical impression that extraocular muscle akinesia was not greatly enhanced by its use, while the duration of effect appeared much shortened and orbital edema seemed more prone to occur, when compared with injection of the same volume of solution not containing hyaluronidase. This, however, is a personal impression and is not shared by all members of the Wilmer staff.

### A. Use of Xylocaine

Xylocaine, a comparatively new local anesthetic agent, was introduced in 1946.[7] There now are well over a hundred reports, uniformly enthusiastic, of its use in general surgery and dentistry. However, only one paper has appeared concerning its use for retrobulbar anesthesia in ophthalmic surgery: Doret,[8] a Swiss ophthalmologist, has reported a preference for Xylocaine (two percent). In addition, three authors have claimed Xylocaine is an excellent topical anesthetic for use in the eye.[8, 9, 10]

Xylocaine is very stable. It is not decomposed after boiling with 30-percent hydrochloric acid for eight hours or lengthy heating with strong alcoholic potassium hydroxide. Xylocaine hydrochloride is soluble at a pH of 6.8 or below.

Xylocaine by injection is reported to spread better than, act faster than, give deeper anesthesia than, and last from two to two and one-half times as long as procaine. The addition of epinephrine is said to approximately double the duration of each.[7]

The toxicity of Xylocaine is quantitatively close to that of procaine, probably very slightly greater. A recent paper by Southworth and Dabbs[11] reports of 68,281 cases collected from the literature, with only four instances of significant but nonfatal reactions, even though total doses of 3.0 gm. or more had been used in some instances.

More than a year ago, we occasionally began trying Xylocaine, without any real enthusiasm, from samples of two-percent solution which had been sent us. In the course of time, we became more and more impressed with its comparative effects. We then obtained solutions of Xylocaine in concentrations of two, three, four, and eight percent. By trial, we decided the four-percent solution (with adrenalin added) was the strongest concentration which did not tend to cause orbital edema. This may possibly be explained by the fact that the four-percent solution is equivalent in molarity to normal saline.

We gradually became sufficiently impressed with Xylocaine to undertake an objective evaluation of its effectiveness, as compared with procaine, for retrobulbar injection.

In order to make such a comparison, the residual motion in each of the extraocular muscles was graded 15 minutes after injection of 1.5 to 2.0 cc. of one of the following solutions:

*A.* Procaine (four percent) containing adrenalin (1:20,000).

*B.* Procaine (four percent) containing adrenalin (1:20,000) and hyaluronidase (5.0 to 15 u./cc.

*C.* Xylocaine (four percent) containing adrenalin (1:20,000).

To eliminate any subjective element, the assistant selected the solution for individual cases in a more or less rotational but partially random sequence. The surgeon, without knowing the solution used, graded the approximate percentage residual motion in each muscle. The injections were given by the assistant, using a 1.5-inch, 22-gauge needle, through the temporal portion of the lower lid, aimed toward the center of the muscle cone, with the eye turned up and slightly nasally. For certain cases, such as corneal transplant or retinal detachment operation, the surgeon requested that Xylocaine be used, but these

cases were not included because of the possible subjective influence on grading.

After 17 cases of each had been graded, use of solution *B* (containing procaine, adrenalin, and hyaluronidase) was discontinued for the reason we believed hyaluronidase resulted in shorter duration of anesthesia and occasional orbital edema, without markedly better akinesia.

During the past several weeks, after the data concerning the solutions of procaine (four percent), procaine (four percent) with hyaluronidase, and Xylocaine (four percent) had been tabulated, we have injected a fourth combination (solution *D:* Xylocaine (four percent) containing adrenalin (1:20,000) and hyaluronidase (5.0 to 15 u./cc.) in a series of 20 consecutive cases. Statistical comparison of this last group with the preceding groups is not strictly valid because of the following factors:

1. Possible subjective bias.
2. Consistent use of 2.0 cc., followed by five minutes' pressure on the lids, whereas, the other injections had varied from 1.5 to 2.0 cc., and pressure had been applied only if the orbit had a tendency to be "tight."
3. Possibly more accurate placement of the injections because of greater experience of the assistant.

## Results

We could not time the duration of anesthesia or akinesia consistently without some danger to the operated eye. Our estimations of duration, therefore, are based largely on cases where surgery lasted long enough for some effect to wear off. Table 1 illustrates the longer effect of Xylocaine.

TABLE 2

AKINESIA OF THE EXTRAOCULAR MUSCLES

| Retrobulbar Injection of 1.5 to 2.0 cc. Solution Containing Adrenalin, 1:20,000, plus: | No of Cases | Average Residual Motion (percent) |
|---|---|---|
| A. Procaine (4%) | 45 | 15.4 |
| B. Procaine (4%); Wydase (5–15 u./cc.) | 17 | 14.5 |
| C. Xylocaine (4%) | 38 | 10.2 |
| D. Xylocaine (4%); Wydase (5–15 u./cc.) | 20 | 3.2 |

The more profound effect of Xylocaine on depth of motor-nerve akinesia is brought out in Table 2, which shows the average residual extraocular muscle actions after injecting each of the four solutions tested. Statistically, the superiority of solution *C* over *A* would occur as a result of sampling error only one time out of eight, and the indicated superiority of solution *D* over solutions *A* or *B* would have no real possibility of being due to chance occurrence, except that the factors mentioned above could have influenced the comparative results.

The known fact that effects of local anesthetics vary inversely with the diameter of the nerve fibers is borne out by our observations. The depth of anesthesia with Xylocaine, with or without hyaluronidase, consistently has appeared greater than with procaine, while the complete freedom from pain with Xylocaine during operations on acute or chronically inflamed eyes has been most impressive.

Premedication with nembutal and seconal made it impossible to collect completely relia-

TABLE 1

DURATION OF EFFECT
(Approximate averages in minutes)

| Retrobulbar Injection of 1.5 to 2.0 cc. Solution Containing Adrenalin, 1:20,000, plus: | Complete Sensory Anesthesia | Nearly Maximum Akinesia of Extraocular Muscles |
|---|---|---|
| A. Procaine (4%) | 75 | 40 |
| B. Procaine (4%); Wydase (10–20 u./cc.) | 50 | 30 |
| C. Xylocaine (4%) | 180 | 90 |
| D. Xylocaine (4%); Wydase (10–20 u./cc.) | 120 | 60 |

TABLE 3

TEMPORARY ANESTHESIA OF OPTIC NERVE

| Retrobulbar Injection of 1.5 to 2.0 cc. Solution Containing Adrenalin, 1:20,000, plus: | Approximate Incidence of Transient Loss of Nearly All Light Perception (percent) |
|---|---|
| A. Procaine (4%) | 7 |
| B. Procaine (4%); Wydase (5–15 u./cc.) | 15 |
| C. Xylocaine (4%) | 25 |
| D. Xylocaine (4%); Wydase (5–15 u./cc.) | 75 |

TABLE 4

AVERAGE RESIDUAL MOTION OF THE INDIVIDUAL EXTRAOCULAR MUSCLES

All 124 cases: Solutions A, B, C, or D

| | Percent |
|---|---|
| Superior oblique | 15.7 |
| Lateral rectus | 15.5 |
| Superior rectus | 13.9 |
| Lateral rectus | 10.9 |
| Inferior rectus | 7.6 |
| Inferior oblique | 3.6 |

ble data regarding anesthesia of the optic nerve, but Table 3 gives our best estimate of the percentage incidence of temporary loss of all or almost all light perception following the various injections.

Table 4 is inserted merely to record the relative difficulty of paralyzing the various extraocular muscles by retrobulbar injections such as we used, as revealed by the over-all averages from all four of the solutions tested.

We have found the use of 2.0 cc. of Xylocaine (four percent) containing adrenalin (1:20,000), with or without hyaluronidase, almost always gives a complete seventh-nerve block, and that such an O'Brien block can be counted on to remain complete far longer than is the case with procaine.

### TOPICAL

Bjork[9] found that topical instillations of Xylocaine had no effect on the pupil or the blood vessels of the conjunctiva, and he states four-percent Xylocaine solution compares in anesthetic quality with two-percent cocaine, while six-percent Xylocaine compares with four-percent cocaine.

We have used four- and six-percent Xylocaine topically. Both produce fairly good corneal anesthesia. The four-percent solution is almost isotonic with normal saline, while the hypertonic six-percent solution approximates the tonicity of tears. The latter has appeared to be useful in retinal detachment surgery, where continued clarity of the cornea is important.

### SUMMARY AND CONCLUSIONS

A comparison has been made of the effects of the retrobulbar injections of 1.5 to 2.0 cc. of the following solutions: *A.* Procaine (four percent) containing adrenalin (1:20,000). *B.* Procaine (four percent) containing adrenalin (1:20,000) and hyaluronidase (5.0 to 15 u./cc.). *C.* Xylocaine (four percent) containing adrenalin (1:20,000). *D.* Xylocaine (four percent) containing adrenalin (1:20,000) and hyaluronidase (5.0 to 15 u./cc.).

The following conclusions have been reached:

1. The effect of Xylocaine on both sensory and motor nerves is more profound than, and lasts over twice as long as, the effects of procaine on these nerves.

2. The duration of either is increased by adrenalin, but diminished by hyaluronidase.

3. Although hyaluronidase added to either procaine or Xylocaine probably gives a more consistently complete block, it is questionable whether inclusion of this enzyme is desirable because of the shorter effect and occasional annoying orbital edema which attend its use.

*429 Medical Arts Building,*
*The Henry Ford Hospital (2).*

### REFERENCES

1. Koller, K. (translated by H. Knapp): Arch. Ophth., **13**:402, 1884.
2. Knapp, H.: Further observations on the use of cocaine. Med. Record, **26**:656, 1884.

3. Goodman, L., and Gilman, A.: The Pharmacological Basis of Therapeutics. New York, Macmillan, 1941.
4. Atkinson, W. S.: Use of hyaluronidase with local anesthesia in ophthalmology. Arch. Ophth., **42**: 628, 1949.
5. Lebensohn, J. E.: Hyaluronidase in ocular surgery and therapy. Am. J. Ophth., **33**:865, 1950.
6. Tassman, I. S.: The use of hyaluronidase in ophthalmology. Am. J. Ophth., **35**:683, 1952.
7. Lofgren, N.: Studies on Local Anesthetics: Xylocaine, A New Synthetic Drug. Stockholm, 1948.
8. Doret, M.: La Xylocaine en ophthalmologie. Praxis, **39**:491, 1950.
9. Bjork, A.: On possibility of testing the surface anesthetic effect of local anesthetics on the human cornea: Various viewpoints on surface anesthesia in ophthalmologic practice. Acta Ophth., **28**:229, 1950.
10. Offret, G., and Decaudin, A.: Premiers essais en France d'un Nouvel anesthesique. Bull. Soc. franç. d'ophtal., 1950, p. 64.
11. Southworth, J. L., and Dabbs, C. H.: Xylocaine: A superior agent for conduction anesthesia. Current Res. Anesth., **32**:159, 1953.

# USE OF PARENTERAL TRYPSIN IN ACUTE INFLAMMATORY AND THROMBOTIC OCULAR DISTURBANCES*

JOSEPH M. HOPEN, M.D.
*Philadelphia, Pennsylvania*

## INTRODUCTION

When enzymatically induced anticoagulation, thrombolysis, and fibrinolysis followed intravenous administration of purified crystalline trypsin to 60 rabbits and 25 dogs, it seemed that intravenous trypsin might significantly alter physiologic processes characterized by the development of coagulated plasma, vascular thrombi, and deposition of fibrin. It also suggested that trypsin initiates biochemical reactions which might result in prompt and sustained subsidence of acute inflammation. Subsequent clinical studies, based on 6,456 trypsin infusions to 538 patients, gave evidence that trypsin rapidly suppresses acute inflammations of diversified origin (bacterial, viral, allergic, and chemical).[1-4]

The purpose of this report is to document satisfactory results when parenteral trypsin was given in 11 of 17 ocular cases refractory to cortisone. Results in seven of the 11 cases were impressive. Complete recovery took place in four of six cases in which intramuscular trypsin was instituted. Dramatic subsidence of all signs and symptoms of acute inflammation occurred in one of two select cases in which subconjunctival injections of trypsin were attempted in conjunction with courses of intravenous trypsin.

## CASE REPORTS

Parenteral trypsin was administered to these patients during the course of acute inflammatory and thrombotic ocular processes. The diagnoses are shown in Table 1.

## METHODS AND PROCEDURE

One vial of purified crystalline trypsin† specially prepared for intravenous use, containing 10 mg., was dissolved in 100 cc. of isotonic saline solution. A typical course of therapy consisted of two infusions daily for six consecutive days. The rate of intravenous administration of trypsin did not exceed 30 drops per minute. There was an interval of three to five hours between infusions of trypsin, during which time a slow continuous intravenous infusion of five-percent glucose in saline was continued, thus keeping the vein patent for the second infusion.

A typical course of intramuscular therapy consisted of injections of four mg. of trypsin, twice daily for six days. The injection was given deep in the gluteal muscle.

Ten mg. of trypsin (0.5 cc. in solution)

* From the Philadelphia General Hospital.

† The trypsin used in the ophthalmic cases was supplied by the National Drug Company.

TABLE 1

USE OF TRYPSIN IN OCULAR DISTURBANCES

| Eye Disease | Number of Patients Given By Intravenous | Intramuscular | Subconjunctival |
|---|---|---|---|
| Panophthalmitis | 1 | | |
| Acute iritis | 4 | 4 | |
| Corneal ulcers* | 2 | 1 | 1 |
| Hyphema* | 1 | 1 | 1 |
| Uveitis with secondary glaucoma | 2 | | |
| Central retinal vein thrombosis | 1 | | |
| Totals | 11 | 6 | 2 |

* Subconjunctival trypsin was used in conjunction with intravenous trypsin.

was injected subconjunctivally with an equal quantity of one-percent Pontocaine (0.5 cc.) in the same syringe once daily for three days.

### CLINICAL RESULTS[5]

#### 1. PANOPHTHALMITIS

After intravenous trypsin had been started, there was a diminution in the redness, swelling, and pain in the affected eye. The corneal ulcer which seemed about to perforate, did not. Although the eye itself ultimately degenerated and became soft, it was not necessary to perform an enucleation.

#### 2. ACUTE IRITIS

Four patients with acute iritis were each given a trial of seven days of cortisone and a cycloplegic, with no change noted. However, after discontinuing this medication and instituting intravenous trypsin, there was an immediate subsidence of conjunctival injection, diminution of swelling and pain, decrease in number of cells and decrease in flare in the anterior chamber. Complete recovery took place following the sixth day of infusion of trypsin.

Identical results were apparent with the full course of intramuscular trypsin.

#### 3. CORNEAL ULCERS

In the two cases treated with a full six-day course of intravenous trypsin, only questionable improvement was noted.

In the case treated with intramuscular trypsin no improvement was recorded.

#### 4. HYPHEMA

Postoperative hyphema cleared by the sixth day of the intravenous infusion of trypsin. There was no improvement in the case in which intramuscular trypsin was used.

#### 5. UVEITIS WITH SECONDARY GLAUCOMA

In the two cases treated, both showed favorable subjective response, with notable subsidence of acute pain and headache. Objectively, however, only one case showed a decrease in intraocular pressure and diminution of cells and flare in anterior chamber.

#### 6. CENTRAL RETINAL VEIN THROMBOSIS

In the one case reported, no striking change was noted in vision, other than gradual but very slight improvement to the point of being able to detect gross outlines within a room and to outline lettering on a hospital towel. Objective retinal findings showed very few changes during hospitalization.

### CONTRAINDICATIONS

Those diseases characterized by any bleeding tendency, liver failure, or congestive heart failure should not be treated with parenteral trypsin.

### SIDE EFFECTS

In only one case of the 17, was there any

evidence of untoward effects. This patient had a transitory chill which responded favorably to routine nursing procedures. Intravenous trypsin was resumed the following day.

### Case reports

#### 1. Panophthalmitis

H. H., a 72-year-old white man, had an uneventful intracapsular cataract extraction. Two days postoperatively, upon redressing the operative site, a small amount of green pyogenic material was present in the eye. Bacteriologic examination showed the presence of *B. pyocyaneus*.

The patient was put on large doses of systemic and local antibiotics plus local polysporin ointment. After seven days of this intensive therapy, a corneal ulcer threatened to perforate. All medication was stopped and intravenous trypsin was instituted.

After the fifth day, there was considerable subjective and objective improvement. Examination of the eye at this time revealed a contracted, soft globe, healed corneal ulcer, diminution of all redness, pain, and edema, and absence of purulent discharge. The treatment was terminated at this time.

The eye is blind and phthisical but there is no evidence of recurrence of the infection.

#### 2. Acute iritis

B. S., aged 50 years, developed an acute iritis in the left eye which was treated unsuccessfully with one-percent atropine and cortisone (1:4) for seven days. All treatment was stopped and intravenous trypsin was started.

By the third day there was a marked diminution in pain, lacrimation, and photophobia and a noticeable decrease in the circumcorneal injection. The flare and cells in the anterior chamber had subsided to a considerable degree.

By the end of the course of trypsin, there was only a grade-I flare. An uneventful recovery ensued. Six months' follow-up did not show any recurrence.

#### 3. Corneal ulcer

W. J., aged 75 years, developed a deep corneal ulcer which involved the epithelium, Bowman's membrane, and connective tissue of the left eye. Bacteriologic studies failed to reveal any causative organisms. The eye did not respond to vigorous local and systemic antibiotic therapy.

After the ninth day of unsuccessful therapy, intravenous trypsin was started. The condition of the eye did not improve appreciably.

In an attempt to prevent the ulcer from perforating, a subconjunctival injection of trypsin (10 mg.) was given. This caused much subjective burning and pain. For the second subconjunctival injection, the trysin was mixed with an equal amount of one-percent Pontocaine and the patient felt no burning or pain.

After the third subconjunctival injection, the ulcer appeared to be subsiding. Monthly follow-ups show complete healing of the ulcer with no subjective complaints.

#### 4. Hyphema

L. J., a 51-year-old insulin-requiring diabetic, developed a hyphema in the anterior chamber of the left eye following an iridencleisis. A six-day course of intravenous trypsin seemed to clear the anterior chamber. In addition to the intravenous trypsin, three subconjunctival injections of 10 mg. of trypsin with equal quantities of one-percent Pontocaine were used. In regular monthly check-ups, the anterior chamber of the left eye has remained clear.

#### 5. Uveitis with secondary glaucoma

E. W., aged 47 years, developed a secondary glaucoma in her left eye due to an active iritis. Tension was recorded at 35 mm. Hg (Schiøtz). Signs and symptoms included corneal edema, circumcorneal injection, severe burning pain, and headache, marked lacrimation, and photophobia.

A course of 10-percent neosynephrine and

cortisone (1:4) failed to bring improvement. After eight days intravenous trypsin was substituted for the previous therapy. The corneal edema subsided and the cornea became clear. There was marked diminution of the pain, headache, photophobia, and lacrimation. The anterior chamber was visualized and only revealed grade-II flare. The intraocular pressure was 20 mm. Hg (Schiøtz). The patient failed to return after discharge from the hospital.

6. Central retinal vein thrombosis

E. R., aged 74 years, was admitted to the hospital because blindness suddenly developed in the right eye. Funduscopic examination revealed retinal hemorrhages and venous thrombosis of the retinal veins. Tension was 20 mm. Hg in the right eye and 65 mm. Hg (Schiøtz) in the left eye. The left eye was blind. The right eye had light perception only and a purple color.

Intravenous trypsin was given over a five-day period. No striking change was noted in vision except that the patient was able to detect gross outlines within the room and outline lettering on hospital towel. No significant fundus changes were noted in the left eye.

## Discussion

There was a dramatic subsidence of all signs of acute inflammation in 11 of 17 patients. Trypsin was used subconjunctivally with one-percent pontocaine in two patients without any apparent untoward ocular effects.

When more commonly used drugs appear to have no effect on the pathologic condition, trypsin seems to be an excellent adjuvant therapeutic agent. The proteolytic, fibrinolytic, and anticoagulant properties of trypsin need further clinical trial.

Further investigations will be directed toward the following eye conditions: (1) Uveal tract inflammation of all types, (2) central retinal vein thrombosis, (3) central retinal arterial embolism, (4) hyphema and vitreous hemorrhage, (5) secondary glaucoma due to inflammatory and vascular causes, and (6) corneal ulcers of all types.

## Summary

Trypsin initiates biochemical reactions which result in prompt and sustained subsidence of acute inflammation. Thrombolytic changes occur later. Eleven of the 17 selected cases showed recovery from pathologic ocular conditions. Subconjunctival crystalline trypsin may have a definite clinical value in intraocular hemorrhage. The results in these cases, in which there were no adverse reactions, should encourage further clinical studies.

*2034 Pine Street (3).*

## References

1. Innerfield, I., Schwarz, A., and Angrist, A.: Fibrinolytic and anticoagulant effects of intravenous crystalline trypsin. Bull. N. Y. Acad. Med., **28**:537 (Aug.) 1952.

2. ———: Intravenous trypsin: Its anticoagulant, fibrinolytic, and thrombolytic effects. J. Clin. Invest., **31**:1049-1055 (Dec.) 1952.

3. Innerfield, I., Angrist, A., Schwarz, A., and Ruggiero, W.: Intravenous trypsin: Its effects upon intravascular thrombi and the acute inflammatory reaction. Surgical Forum, Philadelphia, Saunders, 1952, pp. 526-530.

4. Innerfield, I., Angrist, A., and Schwarz, A.: Parenteral trypsin: Clinical application in 528 patients. J.A.M.A., **152**:597-604 (June) 1953.

5. Hopen, J. M.: Use of intravenous and subconjunctival trypsin in acute inflammatory and hemorrhagic ocular disturbances: A preliminary report. J. Philadelphia Gen. Hosp., **4**:89-92 (Sept.) 1953.

# CLINICAL PATHOLOGIC CONFERENCE

PARKER HEATH, M.D.
*Sullivan Harbor, Maine*

## CASE E-53-389

### HISTORY

The patient, a woman, aged 47 years, gave as her principal complaint a painless swelling of the right upper lid associated with slight protrusion of the eyeball. Duration was 10 days but she had had some subjective discomfort in the right orbit for about two months.

### EXAMINATION

A swelling of soft density was seen in the midde and extending over to the upper outer aspect of the right eyelid. Deeper palpation elicited two and possibly three nodules which were movable and presented vague edges. No swelling was noticed or palpable in the pre-auricular node or other lymph nodes. A questionable swelling and firmness was palpable in the submaxillaries on this right side. Ptosis, apparently mechanical, caused a drooping of the right upper lid so that the palpebral fissure was about one-half closed.

Inspection of the conjunctiva showed only a slight chemosis. No discharge was noted. Exophthalmic measurements (Hertel) were: R.E., 24 mm.; L.E., 20 mm. The ranges of the extraocular movements were possibly slightly limited in the upper gaze by the right eye, and normal in the left eye. Vision measurements were: R.E., 20/25; L.E., 20/20. Ophthalmoscopic examination showed a slight congestion and increase in caliber of the retinal veins in the right eye; the left eye appeared normal.

X-ray examinations of the orbit showed slight, increased tissue density in its upper aspect without any bony changes.

An operation was done.

### DIFFERENTIAL DIAGNOSIS

*Discussion by Dr. Benjamin Sachs**

We are confronted with the problem of a differential diagnosis when the patient has a unilateral exophthalmos, partial ptosis, accompanied by a palpable mass or masses in upper orbit. In this case exophthalmos was moderate and the patient had questionable unilateral disturbance of ocular motility in the upper gaze. Nothing is said about the state of this patient's metabolism or the condition of her neck and we assume, therefore, that these examinations would give normal findings. Genetic disease and orbital anomalies can probably be eliminated.

Inflammations of the orbit, that is, orbital cellulitis—particularly the granulomatous nodular forms—must be considered. These can arise from sinuses, walls of orbit, extraocular muscle, lacrimal apparatus, or foreign bodies. Some granulomas, such as the eosinophilic granulomas, are very obscure in origin. These attack bone in the early stage.

Infections of skin of the face have been known to produce orbital cellulitis and exophthalmos. The exophthalmos usually is a comparatively severe acute process, taking a different course from that indicated in this patient.

The so-called myositis, more often than not, is an orbital cellulitis with involvement of the extraocular muscles. This is a fairly chronic nodular granulomatous process with varying degrees of exophthalmos. The extraocular muscles show limitations, however, which is not a prominent feature in this case. The absence of fever and sickness in general is against a subacute orbital cellulitis.

Affections of the lacrimal apparatus could be causative factors in limited exophthalmos but they are characterized by disturbances in the inner aspect of the orbit not seen in this case. No history is given relevant to a foreign body or trauma. Some forms of tenonitis are unilateral but rarely show the nodule relations described in the eyelids of this case.

One should review the possibilities of an

* Surgeon in Ophthalmology, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.

intraocular origin for orbital disease, including extending tumors. Ophthalmoscopic examination, however, was relatively normal. We can therefore rule out intraocular neoplasm.

An obscure metabolic disease of the orbit related to some endocrine etiology—pituitary, thyroid, or adrenal in origin—is not likely from the history. Nervous origins of orbital disease such as sympathetic paralysis are not suggested by the admitted findings. No pupillary disturbances are given; an apex syndrome is not suggested by trauma or X-ray findings, and we can discard this thought.

A first consideration of tumor suggests the most common tumor of the orbit, namely, hemangioma. Appearance, findings by palpation, age of the patient, absence of known angiomatous changes probably can rule this out.

A lipoma is uncommon and does not appear at the sites indicated by examination. Furthermore, these tumors are, more often than not, bilateral, although unilateral protrusion of fatty material through the orbital septum does appear as early as the age of 47 years.

Lymphoma is a possibility. The multiple nodules are somewhat inconsistent with the usual lymphoma. Nothing is said regarding any systemic vascular dyscrasia, consequently we probably can rule out lymphoma.

Meningioma is a possibility to be considered, although unlikely in view of the X-ray findings.

A prime consideration is a tumor emanating from the lacrimal gland. The growth described in the protocol, however, is nearer the midline and has already somewhat affected upper gaze. It is dense enough to show on the X-ray film. The patient's age is somewhat against this tumor, therefore, this diagnosis is laid aside as a possibility.

The last, and what seems to me to be the most important possibility, is orbital granuloma—so-called pseudotumor. Gradual onset, physical findings, X-ray results, and course, as indicated in the short history, are compatible. So-called pseudotumor might better be called orbital granuloma or nodular orbital inflammation. A person of any age may be, but the adult is more likely to be, attacked.

The inflammatory mass can be unilateral or bilateral and begins with a stormy or a gradual and mild onset. Disturbances of the ocular motility and some exophthalmos are characteristic. The inflammation creates a space-taking mass in the orbit, bringing about exophthalmos and compression of the veins and thus, in turn, congestion in the fundus and chemosis in the conjunctiva.

Sarcoidosis with a long course and with periods of abeyance involves the lacrimal glands of some patients. Other attacks by this disease on other parts of the body are quite variable and may not be noticed clinically. The lacrimal-salivary involvement is called Mikulicz's disease. No other evidence is given in the protocol for sarcoidosis.

## Diagnoses

*Clinical diagnosis.* Possible orbital tumor.

*Dr. Sach's diagnosis.* Possible orbital granuloma (pseudotumor).

*Pathologic diagnosis.* Dacryadenitis, orbital granuloma (Mikulicz's).

## Comment

### *Dr. Parker Heath*

This patient has no history of keratoconjunctivitis sicca. She further had no history of rheumatoid joint involvement. As a generality, the lacrimal gland is subject to inflammations from various agents. It reacts somewhat nonspecifically by edema, proliferations of ductal epithelium, accumulations of lymphocytes by fibrosis, and, in later stages, fibrotic atrophy. The accessory lacrimal gland tissue in the eyelids can become involved similarly. It is the combination involvement of lacrimal and salivary or parotid glands which has led to the Mikulicz's designation.

Sections made from the biopsy material of the case herein reported show low-grade, chronic, granulomatous inflammation with fibrous repair and replacement.

# NOTES, CASES, INSTRUMENTS

## MAGNETIC FOREIGN BODY

### Report of an intraocular case in which removal was successful after 24 years

Gregory Flynn, M.D., and Morgan Raiford, M.D.
*Atlanta, Georgia*

The intolerance of the eye to iron foreign bodies is well known. A report of the following case seems justified because of the unusually long period (24 years) the foreign body was retained without the usual serious complications of siderosis.

### Case history

The patient was a 29-year-old white man who was first seen on September 15, 1952, with a chief complaint of periodic redness and irritation of the left eye. He gave a history of a penetrating wound of the left eye by a steel fragment 24 years ago. The ophthalmologist consulted at that time made no attempt to remove the fragment.

Vision was reduced to light perception after the injury. The eye remained quiet until 1946 when intermittent episodes of redness and irritability occurred. At that time, the patient was stationed in Germany and had completed two years of combat (Infantry). He received an ordinary discharge.

He was relatively asymptomatic until 15 months ago, when episodes of irritability and transient blindness occurred involving both eyes. They lasted 10 to 15 minutes and were followed by severe headaches. The time interval between these episodes diminished from months to weeks. He then consulted an ophthalmologist, who recommended enucleation. We were independently consulted for corroboration.

*Examination* revealed a foreign body partially imbedded in the lens and projecting into the anterior chamber with rust staining of the surrounding cataractous lens and iris (fig. 1). Tension was 16 mm. Hg (Schiøtz),

Fig. 1 (Flynn and Raiford). Preoperative appearance, showing intraocular foreign body.

O.U. Except for peripheral anterior synechias in the upper nasal quadrant, the angle was normal. Light projection was good. Two-point light discrimination was less than one cm. at one m. No flare was seen. Examination of the right eye was essentially negative. He also had a left exotropia of 45 degrees (Hirshberg).

On September 17, 1952, a foreign body measuring 3 mm. by 1 mm. by 1 mm. was easily removed with a magnet through a small limbal incision. Capsulectomy followed by irrigation was carried out without complications. The postoperative course was uneventful (fig. 2).

On December 15, 1952, visual acuity was 20/30 with a +3.25D. sph. ⌒ +1.5D. cyl.

Fig. 2 (Flynn and Raiford). Postoperative appearance, after surgical removal of the foreign body and iridectomy.

ax. 45°. There was no evidence of siderosis on funduscopic examination. The fields were normal. On March 4, 1953, a six-mm. recession of the left lateral rectus muscle and a 10-mm. resection of the left medial rectus muscle were done under local anesthesia. On March 29, 1953, the patient had second-degree fusion at minus-four prism diopters on the troboscope with the subjective and objective angles equal. He was given the following correction: O.D., +0.5D. sph. ⊂ +0.25D. cyl. ax. 105° = 20/20; O.S., +3.25D. sph. ⊂ +1.5D. cyl. ax. 45° = 20/30. Add: O.S., +1.25D. sph. and +2.25D. sph.

The patient was unable to take time from his work for orthoptic training. When last seen on May 15, 1953, he was suppressing O.S., and had stopped wearing his prescription. This was not unexpected but the surprisingly small anisometropia seemed to justify a trial.

### Discussion

The rapidity of development of siderosis bulbi is probably dependent on the location of the intraocular ferrous body. In the case reported, the fragment was exposed to the aqueous currents which presumably carried the slowly oxidized particles from the eye via Schlemm's canal. This may have prevented any significant concentration of the irritating substance in the eye. It is very possible that the metal was completely buried in the lens until recent years when the lens matter may have eroded about the particle causing the onset of symptoms.

The episodes of blindness affecting the uninvolved eye are difficult to evaluate. Their briefness would suggest either an angiospastic etiology or hysteria. The latter seems inconsistent with the patient's psychologic make-up.

### Summary

A patient is presented with a 24-year history of a well-tolerated intraocular magnetic foreign body. This was surgically removed with restoration of potentially useful vision.

### Conclusions

1. Magnetic intraocular foreign bodies may be present for years without the development of siderosis bulbi.

2. Enucleation should never be considered until a careful examination has ruled out all possibility of alternative treatment.

*144 Ponce de Leon Avenue, N.E.*

### References

von Hippel, E.: Ueber Siderosis bulbi und die Beziehungen zwischen siderotischer und hamatogener Pigmentierung. Arch. f. Ophth., **40**:123, 1894.

de Schweinitz, G. E.: Diseases of the Eye. Philadelphia, Saunders, 1921, ed. 9, p. 623.

Wolff, E.: Pathology of the Eye. Philadelphia, Blakiston, 1948, pp. 177-179.

Wurdemann, H. V.: Injury of the Eye. Chicago, Cleveland Press, 1912, p. 365.

---

## ORBITAL RETRACTOR

Robert L. Alexander, M.D.
*Sacramento, California*
and
Peter H. Ballen, M.D.
*Hempstead, New York*

A retractor for exposure of the deeper structures of the orbit and deeper portion of eye muscles is not available. The Army right-angle retractor was not originally intended for eye work and, as a result, is clumsy and not adaptable. The Arruga type of retractor is not satisfactory for many exposures.

A retractor was needed: to expose deeper portions of muscles, to keep extraneous material away from the site of operation, to permit passing of a needle toward the eyeball without fear of striking the eyeball, to permit a needle deep in the wound to be manipulated without engaging nearby tissues, and yet to keep the assistant's hand out of the operative field. It was felt a curved, solid-blade re-

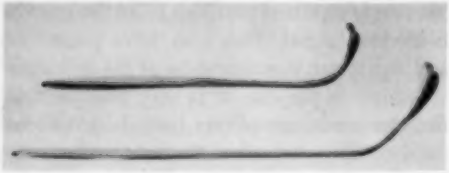


Fig. 1 (Ballen and Alexander). Orbital retractors, lateral view, showing shaft and angulation.

tractor would be most satisfactory.

The two retractors illustrated (fig. 1) were designed* to fulfill these demands. These retractors are made of stainless steel and can be bent to any desired angle or curve in the shank or handle. The long shank of one retractor is designed to use on the nasal and the infra- or supraorbital side of the orbit. The long shank and adjustable angle permit the assistant to place the retractor with easy clearance of the bridge of the nose or the brow, yet permitting the blade to penetrate well into the orbital cavity as needed. The assistant's hands are out of the field of operation and by tilting the handle, the angle of the retractor blade is easily controlled.

The smaller retractor is for the temporal side of the orbit in adults. The angle can be adjusted so that the handle tends to lie back parallel to the side of the head, placing the assistant's hands away from the operative field. It has also been found the retractor of choice in surgery on children because of the smaller blade.

The lip (fig. 2) on the front edge of the blade was placed to secure sure retraction of the tissues. It greatly helps in preventing the frail eye tissues from escaping from under the blade without the operator applying unusual force through the retractor. This lip is excellent in retracting conjunctiva from sclera. Its angle with the blade is just enough to hold gently but firmly when an exposure is made only through the conjunctiva and Tenon's membranes. The retractor saves the

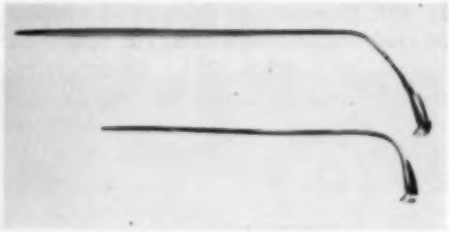


Fig. 2 (Ballen and Alexander). Orbital retractors, showing lip.

trauma of retracting with the crushing pinch of forceps.

The concave surface, deeper at the fore end, is designed to give a greater area of exposure deep in the site of an operation (fig. 3). Straight blades do not permit this exposure.

In addition to serving their original purpose, the retractors can be covered with an insulating lacquer and used for surgery in electric cautery procedures.

The retractors have been found satisfactory in more than 90 successive ocular operations.

Fig. 3 (Ballen and Alexander). Orbital retractors, showing concave surface.

* Original design made and machined with the co-operation of Mr. Samuel Woods, Geneva, New York. Replicas available from Storz Instrument Company, St. Louis, Missouri.

SUMMARY

1. A tissue retractor for general intra-orbital surgery is not available.

2. Two retractors were designed that in principle are similar for orbital retraction.

3. Each retractor has a solid curved blade with a prominent lip and with a shank and handle which can be bent to any desired angle.

*1127 Eleventh Street (14).*

## IMPROVED ORBITAL IMPLANT

H. ARRUGA
*Barcelona, Spain*

In the last few years, there have been many attempts to produce an implant which will assure good prosthetic mobility, but most of these have failed for one or another of the following reasons:

Either the implant was not totally covered by the conjunctiva and intense secretion occurred (this happens when the implant projects through the conjunctiva to fit the artificial eye); or, in the absence of such projection, the mobility is inadequate.

The implant I am describing is a modification of that of Moura Brazil and has now been employed by me in 26 cases with no expulsion or annoying secretion. It is made of acrylic (transpex) and in shape it is three-fourths of a sphere (fig. 1).

The flat anterior side has a central cavity uniting four tunnels for the tendons of the four recti. Two small stainless metal rods project forward from one side of the cavity and articulate with a depression in the back of the artificial eye. The rods are one mm. in diameter and 6, 7, or 8 mm. in length, according to the size of the implant.

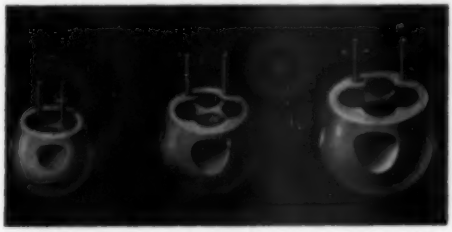


Fig. 1 (Arruga). Improved orbital implants.

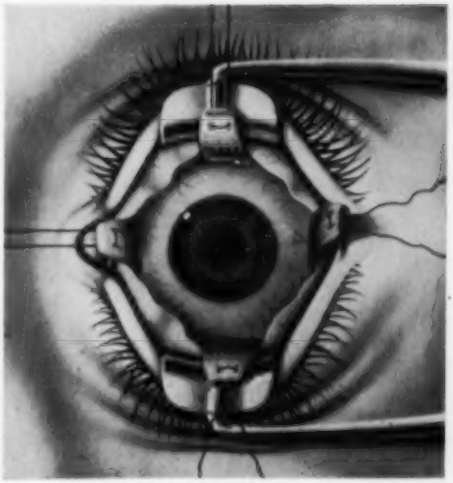


Fig. 2 (Arruga). Disinsertion of the four recti; a suture is passed through each tendon.

The implants are made in three different sizes, 14, 16, or 18 mm. diameter. The first is employed after excision of gliomas; the last in patients with big orbits; the intermediate size suits the majority of cases.

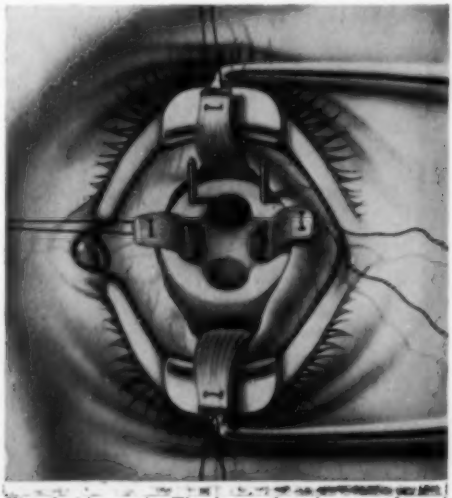


Fig. 3 (Arruga). The internal and external recti are passed through the tunnels of the implant.

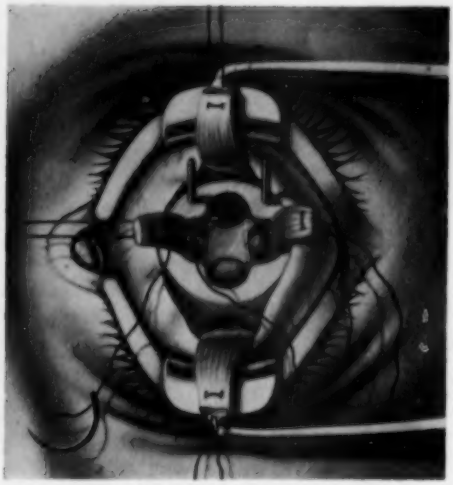


Fig. 4 (Arruga). The suture of the external rectus, which is already knotted, is passed through the internal rectus. The suture through the internal rectus is removed.

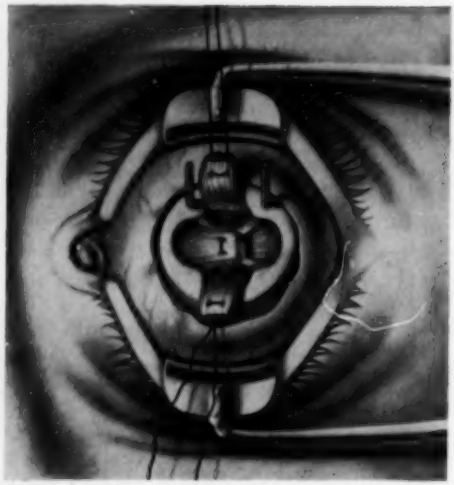


Fig. 6 (Arruga). The superior and inferior recti are passed through the tunnels of the implant.

The fixation of the implant is attained by fusion of the four rectus tendons into a solid cicatricial block at the center of the implant. The absence of secretion is due to the fact that the conjunctiva covers the whole implant after the operation, since the rod is not left in the wound. It is of utmost importance to leave the rods buried initially so that they perforate the conjunctiva spontaneously later. These late perforations never cause secretion and, in some cases, one can even see that the epithelium has grown a little upward covering the base of the rods. If the rods are left between the lids of the conjunc-

Fig. 5 (Arruga). Stage in the operation when the suture has been knotted.

Fig. 7 (Arruga). The suture of the inferior rectus, which is already knotted, is passed through the superior rectus whose suture is removed.

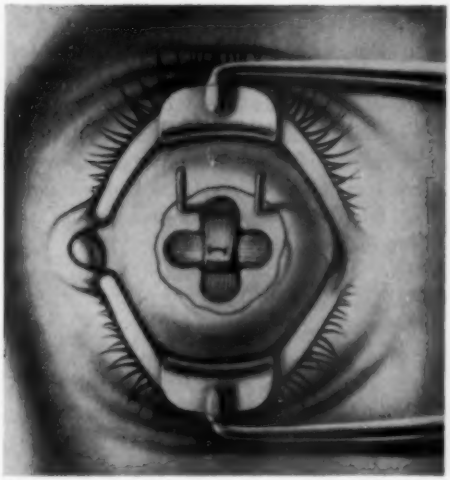


Fig. 8 (Arruga). View of the operating field with the sutures knotted.

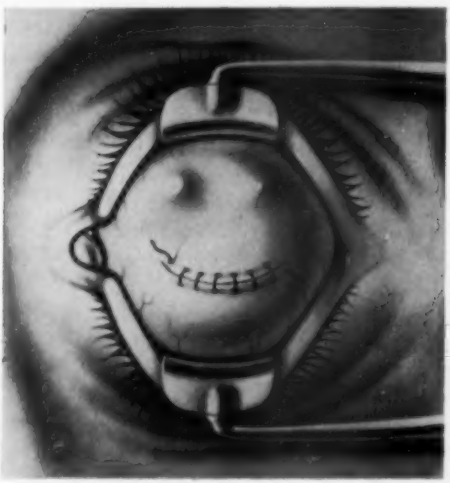


Fig. 9 (Arruga). Sutures of the conjunctiva. The rods are covered by the conjunctiva.

tival wound, the latter does not heal completely and discharge occurs.

Figures 2 to 10 show the different stages of the operation which is extremely simple. The artificial eye should be made of plastic material.

*Pas. Méndez Vigo, 3.*

---

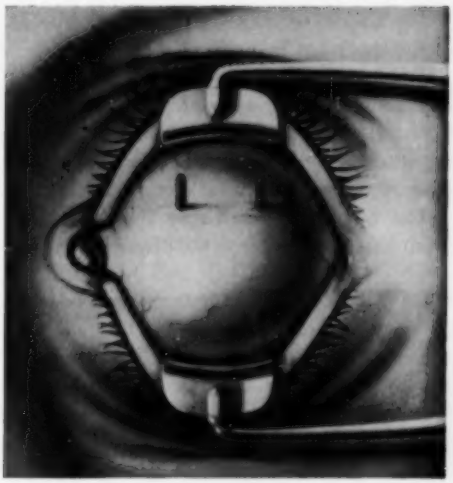


Fig. 10 (Arruga). After a few days, the rods perforate the conjunctiva.

## OFFICE TREATMENT TRAY

RALPH J. CARBO, JR.
*Washington, D.C.*

A small compact unit with the items necessary for office treatment is made of 0.25-inch plywood, 12 by 8 by 5 inches, and is divided into three sections.

Fig. 1 (Carbo). Office treatment tray.

The bottom section has a 11.5 by 7.5 by 3-inch drawer for instruments and loupe.

The middle section has a small shelf, one-inch wide and 1.75-inches high to hold eight 0.5-oz. dropper bottles with various standard medications, kept in place by loops of elastic tape.

The top section is 12 by 8 inches. The front half has cutouts to hold four jars: a six oz. for sterilizing instruments, a four oz. for applicators, a short pound jar for ointments, and the Zeigler wash bottle.

The back half has a 3 by 3 by 3-inch gravity eyepad dispenser, which opens at the side and to which is mounted the tape dispenser. There is a holder for tissues and one, optional, for a four-oz. jar for sterile flats.

*1746 K Street, N.W. (6)*

## Ophthalmic Miniature

Albert P . . . . ke, 18 years old, Swinemündestrasse No. 47, musician. R.E., with −3.50 = Sn. LXX at 14 feet. L.E., fingers at 3 feet. Ophthalmoscopically: Numerous formed vitreous opacities floating back and forth in the liquefied vitreous. The retinal detachment extends from the uppermost periphery to the entrance of the optic nerve, and gravitates down in long folds in the periphery. Corresponding defect in the visual field.

Two knives, 2-3 mm. long and about 1.5 mm. wide at the end of two squint hooks were introduced into the left globe. The negative pole in the vertical meridian and the positive pole in the horizontal as far out in the periphery as possible. The squint hooks were connected with electric wires to a "galvanic" cell. Beginning with one cell (¼ minute duration), then two cells (¼ minute), and three cells (⅓ minute), the total duration of the galvanic current lasted ⅚ of a minute. With the exception of the introduction of the knives, the operation was painless (yellowish light flash), as was the postoperative course. The signs of reaction in the choroid and retina were minimal, and limited to the immediate area of the insertion only. The vitreous apparently was left intact. Already when the dressings were changed for the first time after four days, the retina was in place throughout. The visual field was full. Vision, −4.0 = Sn. L at 14 feet.

Prof. Dr. Schöler,
Klin. Monatsbl. f. Augenh., 1893, p. 215.

# SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

## YALE UNIVERSITY CLINICAL CONFERENCES

March 13 and March 27, 1953

DR. R. M. FASANELLA, *presiding*

### METASTATIC CARCINOMA OF CHOROID

DR. FREDERICK A. WIES, assistant clinical professor of ophthalmology, Yale University School of Medicine, presented two cases of metastatic carcinoma of the choroid. Both cases were of women and metastasis was from the breast. In the first case, in addition to a growth in the choroid, there were extensions into the orbit. This resulted in exposure keratitis, for which a tarsorraphy was performed. This gave the patient relief for the remainder of her life, about two months.

In the second patient, a typical swelling was noted temporal to the disc of the left eye. This patient had had her left breast removed, but repeated X-ray examinations failed to reveal any evidence of metastasis. Many oculists saw this patient, and most of them agreed that the mass was probably metastatic. Finally general metastases developed and the patient died.

### SURGERY OF ENTROPION

DR. WIES then discussed the surgery of entropion, particularly that relating to senile or spastic entropion. He described an operation which has given good results in his hands. This is done as on office procedure under local infiltration anesthesia. The lower lid margin is incised about three mm. below the free border and the incision is carried right down through skin, muscle, tarsus, and conjunctiva. Three double-armed sutures are then placed in the lower lip of the conjunctiva and brought through the incision and out the upper border of the skin incision and tied. Several small sutures are placed through the skin between these master sutures. The sutures are all of 4.0 black silk. About 10 days later, the sutures are removed. The resulting scar is hardly visible and no recurrences of entropion have been noted over a four-year period.

*Discussion.* DR. VAN HUEVEN: Is this entropion procedure satisfactory in the usual case?

DR. WIES: This operation doesn't seem to be physiologic but in some cases there have been very good results. One case showed slight thickening of the skin in the operative area. Cautery puncture, in my experience, is more valuable for small degrees of ectropion.

DR. GUIDA mentioned a procedure, which has worked well for him, in which the middle section of the orbicularis fibers near the lid margin—the muscle of Riolan—is transected, allowing the fibers to retract.

DR. FASANELLA: Dr. Wies, how does your procedure differ from that of Snellen's suture?

DR. WIES: In Snellen's procedure, the tarsus is bent; whereas, in this procedure, the tarsus, after incision, is hinged and would therefore tend to give a better result.

### RETINAL DETACHMENT

DR. CHARLES A. PERERA, assistant clinical professor of ophthalmology, College of Physicians and Surgeons, Columbia University, presented a series of colored lantern slides of cases of retinal detachment, and discussed the associated clinical and pathologic features.

The varied pathology in retinal detachment was shown by several illustrative cases, including cases following hemorrhage, trauma, a hole in high myopia, scar contraction of the retina, cysts, and so forth. In a case of detachment following metastatic endophthalmitis, the pathologic findings made evident how impossible it would be to improve surgically.

Following intraocular foreign-body removal, the importance of sealing the operative area by diathermy was noted. Occasionally, resulting organized tissue at the site of injury may pull off the retina later. In cases following an old intraocular foreign-body injury, the detachment rarely can be repaired. The visual progress is poor even if the detachment is replaced, especially with any associated siderosis.

Briefly discussed was operation on a "hopeless" case if it were the only eye. Dr. Perera felt operation was justified if there was any chance at all of success. In a case following a retinal cyst, it was demonstrated that the cyst must be evacuated as well as the retina replaced.

A case of hole in the macula brought up operation on retinal holes without detachment. Dr. Perera mentioned cases he has seen develop detachment after a long period of observation. One must try to determine whether the tear is through the entire retinal thickness or not. Especially when there is a detachment in one eye and a tear without detachment in the other, the speaker believed it was wise to seal the tear. There should be 100-percent good visual results in tear without detachment.

Cases were demonstrated of a tear with an operculum. In many cases this is probably due to vitreous adhesion from a previous low-grade choroiditis. Dr. Perera felt that this was the mechanism of a large number of detachments. Adhesions can be visualized often with the ophthalmoscope, or the slit-lamp and Hruby lens.

A friction ring at the ora serrata was shown. It was in a case of long-standing detachment in which a fibrous plaque formed at the border of the detached part of the retina. This frequently occurs at the ora.

Visual prognosis in detachment was discussed. Slides were shown of macular damage in a detachment of four years' standing.

Cysts may develop very rapidly—even within weeks—and, when the macula is detached, poor central vision will usually result, whether or not cystic changes develop.

Occasionally, in a nonprogressive detachment, there may be seen a line of fibrous plaques at the border of the detached area, especially in an inferior detachment. This may be the reason for the failure of the detachment to extend.

A retinal detachment with exudative choroiditis does not respond well, and operation is rarely indicated. An acute choroiditis with considerable reaction was thought less likely to lead to a later detachment than a mild case with small retinal adhesions. In retinitis proliferans, Dr. Perena has yet to see a successful operative result.

The mechanics of operation were considered next. One should not stop with the localization of one hole, but assume there are many, and one must search for them several times, especially after bedrest with both eyes bandaged. The careful examination of the opposite eye was also emphasized. A change in position of the hole after bedrest is common and, therefore, accurate localization at operation is the important factor. Various methods were mentioned. One method of localization is by use of the ophthalmoscope light through the pupil, visualizing the hole, with the assistant marking the transilluminated spot on the globe. A preliminary surface diathermy spot visualized by the ophthalmoscope can be used in flat detachments, or a Walker pin for more bullous types.

Various clinical types of detachment were mentioned:

1. The bullous detachment in the upper temporal area with a small hole is very common. This is often preceded by a small vitreous hemorrhage or opacity, and the history may give a clue to the original site of the detachment. In this type, postoperatively, Dr. Perera does not tip up the head of the bed. He feels that if the retina settles back considerably after a few days of bedrest, the operative prognosis is better than 90 percent.

2. Traumatic detachment is usually, but not always, below and temporal. The larger the hole, the worse the prognosis. Intraocular

hemorrhage, hyphema, or increased tension make the prognosis less favorable. One should not operate when the vitreous is cloudy and when one thinks he sees a detachment. Wait until the vitreous clears, if it does.

3. Extensive flat detachment with disinsertion has a poor visual prognosis when the macula is involved.

4. In a very bullous detachment, inferiorly without a hole, suspect flat malignant melanoma or metastatic carcinoma. If the hole is not seen at first, it may become visible as the retina flattens. If it remains bullous and no hole is found, consider the possibility of fluid withdrawal, with aspiration test for tumor; or you may consider sealing down the whole area.

5. Large tears or disinsertions have a poorer prognosis. It is harder to seal the hole. Here scleral resection may be indicated to reduce the size of the eye and give a wide band of adhesion. Scleral resection, done behind the hole, may be of some advantage. The less the retina reapplies on bedrest, the poorer the prognosis. A large hole, with the retina not reapplying on bedrest, may indicate a 20 to 30-percent favorable prognosis.

6. Hole in the macula has only 30 to 40-percent favorable prognosis. Dr. Perera prefers surface or partial penetrating diathermy for this type of case.

7. Two varieties of detachment in aphakia were differentiated. One is the case that follows cataract extraction where there was vitreous loss at operation. This has a poor prognosis. The other is an ordinary detachment occurring in an aphakic eye, which carries a better prognosis.

*Discussion.* DR. FASANELLA: How long do you keep the patient in bed postoperatively?

DR. PERERA: Generally, I start getting patients up in a week. They are kept fairly quiet, though not immobile, and no sandbags are used. With superior detachments, I keep them quiet a little longer, but I do not tip up the foot of the bed. If the retina is flat at the end of the operation and diathermy coagulation areas are around the tear, the prognosis is good. If the hole is still seen after seven days, the prognosis is poor, unless the hole seems definitely sealed off by diathermy areas around it.

DR. FASANELLA: How do you manage a case of cataract with detachment?

DR. PERERA: The prognosis is poor when the media are cloudy and the exact extent and localization of the detachment is not visible. You may do the cataract extraction first, then, several weeks later, do the detachment. I prefer the intracapsular extraction with complete iridectomy. You are taking a risk whichever you do first, but if it is an uncomplicated cataract, do that first. The detachment may be stationary.

DR. VAN HEUVEN: I agree that it is very important to see what you are doing. In a similar situation I just did a cataract extraction and plan to do the detachment later.

*Question:* Do you use pinhole glasses postoperatively?

DR. PERERA: I use them when patients start getting up. First, with a five-mm. opening, then enlarging it after three or four weeks. I see no point in keeping it up, after that.

DR. VAN HEUVEN: I do not like to let patients read postoperatively.

DR. PERERA: I agree. Reading motion may not be advisable. I do not let them read for three weeks. Television is all right.

DR. ROSENTHAL: Have you used electrolysis?

DR. PERERA: Yes, but the reaction is too small. I do not like it and do not use it now. Also, the amount of diathermy for each patient is an individual thing; a dry sclera is important.

DR. WILLIAM GLASS: Dr. Perera, you mentioned that as the retina settles back, the position of the hole may shift considerably. I find that, at operation for a bullous detachment with considerable retinal elevation, it may be impossible to visualize the preliminary localizing diathermy area through all the subretinal fluid. How do you manage that

type of case? Would you withdraw some fluid first?

DR. PERERA: That is sometimes a problem. What I would do is estimate as well as I can where the hole would be after replacement of the retina, and do the diathermy coagulation accordingly, then release fluid, and check. If the area of the hole has been misjudged, additional diathermy can be done then.

DR. GLASS: How soon would you re-operate when some but not complete reattachment occurs after the first detachment operation?

DR. PERERA: If, after seven days, the hole is still present but seems to be completely surrounded by adequate diathermy, I would wait. However, if still detached and insufficiently sealed by diathermy, the prognosis is poor and re-operation could be done any time, probably after two to three weeks, to let the postoperative reaction subside.

William I. Glass,
*Recording Secretary.*

## MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

May 4, 1953

DR. ROBERT COOPER, *presiding*

### DENDRITIC ULCER WITH DEEP PENETRATION OF CORNEA

DR. WALTER FINK reported the case of a young man who had a corneal ulceration, which began two weeks previously, following the "flu." He had two previous episodes similar to this in the last two years in the same eye but mild in degree.

Examination showed a dendritic type of ulcer involving the outer half of the cornea. The center of the ulcer showed an involvement of Bowman's membrane and the anterior stroma.

Treatment consisted of curettement of the ulcer and the removal of the entire remaining corneal epithelium. The entire cornea was then painted with two-percent iodine and, after five seconds, the iodine was neutralized by four-percent cocaine. Atropine was instilled. A pressure bandage was applied for three days, changing it daily. Following this, cortone solution was used locally four times a day. The ulcer healed, leaving a dense opacity in the involved area.

The case emphasizes the need for early treatment to prevent involvement of the deeper corneal tissue. Involvement of Bowman's membrane and the stroma results in a permanent opacity.

The case also emphasizes the importance of a complete removal of all remaining corneal epithelium when the involved area is extensive. Experience has shown that in severe cases the virus is usually present also in the nonulcerated area and recurrence of the process is likely to take place if the epithelium is not removed in its entirety. Antibiotics, both local and systemically, are not of value because the virus is established in the epithelium and is not reached by the medication.

*Discussion:* DR. F. WIPPERMAN stated that in no case of dendritic ulcer has he found it necessary to denude the epithelium completely by the use of chemical cauterization or curettage. However, in most cases, simply applying iodine at the exact site of the dendritic ulcer is not sufficient. It has proven best to denude the corneal epithelium surrounding the dendritic ulcer. Rather than set any definite limits as to how much of the corneal epithelium should be denuded it is advisable to remove all loose epithelium that easily wipes free with the applicator which contains the chemical agent.

DR. R. HORNS said that he has seen one eye with a dendritic ulcer with complete loss of vision and that was a case in which he felt that the eye was neglected because the lesion was not cauterized with iodine until very late in the course of the disease. It is important that iodine and cocaine be used early in cases of dendritic ulcer. Whenever there is a break in Bowman's membrane, or

when Bowman's membrane is damaged, there will be scarring of the cornea.

Dr. Joseph Garten said that, in his experience with dendritic ulcers, he has found that simple cauterization with tincture of iodine (one-half strength) to the lesion, which stains, often is sufficient to control the infection. He concedes that there are problem cases in which complete removal of the corneal epithelium may be necessary; however, he does not think that this should be routine.

Dr. J. Bushard said that, in the treatment of dendritic keratitis, he likes to use a round wooden toothpick to apply half-strength tincture of iodine. By this method there is mechanical, as well as chemical, removal of all the epithelium of the cornea not firmly adherent to Bowman's membrane. The chemical effect, of course, is neutralized with cocaine.

Dr. V. Linberg wished to emphasize the point that Dr. Bushard just made; namely, that the diseased epithelium is usually loose and can be separated from the underlying cornea by rolling or pushing it off with a tightly wound cotton applicator. This is a good method of determining to what extent and over what areas the iodine cauterization should be carried. In the event that incomplete cauterization of the cornea was carried out, the extent of partial cauterization could thus be determined in a fairly orderly fashion.

### Malignant melanoma

Dr. Walter Fink reported the case of a woman, age 68 years, who came because of an unsightly blind right eye. Examination showed absolute glaucoma with no evidence of irritation. The patient for cosmetic reasons wished it removed.

An evisceration of the globe was performed and a plastic sphere was inserted in the globe. The contents were sent for microscopic study although they did not appear unusual.

The laboratory report returned with a diagnosis of malignant melanoma, which was predominantly of the spindle-B type. A reticulum stain demonstrated an argyprophil fiber content of less than 50 percent. The globe was immediately removed. There were no signs of extension beyond the globe.

The case illustrates the hazard of the evisceration operation even in cases in which there is no suspicion of intraocular tumor. This type of operation offers such good cosmetic results and is so free of postoperative complications some surgeons favor it under proper circumstances.

The case also illustrates the importance of examining the curetted contents of the globe even though it appears grossly normal.

It is of interest in connection with this case to consider the prognosis. As there was no evidence of extension beyond the eyeball, one may speculate on the ultimate outcome. On the basis of a survey of 2,535 cases of malignant melanoma of the choroid and ciliary body made by Wilder and Paul, the following was noted:

Of 479 patients dying with metastasis, approximately 80 percent died within five years following enucleation. Almost 20 percent, however, survived this period, and 3.5 percent lived 10 years or more.

The cell, fiber, and pigment classification of Callender and Wilder are of prognostic significance. From least to most malignant they have listed spindle A, spindle B, fascicular, necrotic, mixed, and epithelioid.

The argyrophil fiber content furnishes an indication of rapidity of growth of a tumor. A heavy fiber content is usually associated with a low degree of malignancy. If the tumor is slow growing, fiber production keeps pace with tumor growth and fibers are able to encompass individual tumor cells, forming a barrier to their escape from the globe and particularly into blood vessels. There is a tendency for the spindle-cell tumors to have a higher fiber content than the mixed and epithelioid types. Among the patients followed 10 years or longer all who had tumors composed entirely or almost entirely

of epithelioid cells died with metastasis. There is an increase in malignancy with increased pigment content.

According to these statistics, the tumor as reported in this case was of the less malignant type, being spindle-B cells, although the fiber content was only 50 percent.

*Discussion.* Dr. F. Wipperman said that it is true that excellent movement is obtained with evisceration and insertion of an implant but he feels equally excellent movement is obtained by the use of the Allen implant. The case reported certainly emphasized why we should routinely send to the laboratory the evisceration scrapings for microscopic study. This point is not too well emphasized in textbooks or classroom lectures.

Dr. Richard Horns said that he thinks that the evisceration operation of the globe is a very good operation in many instances. There is always the danger of an intraocular tumor in any blind eye which cannot be examined with the ophthalmoscope. He does not know what percent of such eyes would have tumors but he would imagine that it would be quite small. He thinks that an evisceration of the globe is probably contraindicated in any case in which the inside of the eye cannot be examined with the ophthalmoscope and is contraindicated when there is any history of injury when sympathetic ophthalmia is a possibility.

Dr. M. McCannel said that, when operating upon a blind glaucomatous eye with enough opacity so he cannot view the fundus, he would lean toward doing the Mules' scleral implant operation for the improved cosmetic result. For those actually having a neoplasm, the majority would be discovered during the curettement portion of the operation. For uveal tissue that appeared normal on removal he would do as Dr. Fink did—have it evaluated by a pathologist. We have been led to believe by previous melanoma statistics that even an enucleation is not proof positive that the patient will be free from later metastatic involvement.

Dr. Joseph Garten said he has seen one case in which a malignant melanoma with extension to the lid and orbit developed about one or two years following such a procedure. He believes that this could develop even with negative biopsy of the contents of the globe at the time of evisceration. In view of this possibility, he would prefer simple enucleation wherever possible, with or without an implant.

Dr. V. Lindberg said he has not done evisceration, but in the last four enucleations he has done, two malignancies were found.

Dr. W. Bushard said he has been very happy with the Allen implant.

### Sympathetic ophthalmia following iridencleisis

Dr. Walter Fink reported the case of a man, aged 62 years, who was operated July 10, 1952, for chronic simple glaucoma of the right eye. An iridencleisis operation was performed. The postoperative period was uneventful and the eye eventually became quiet with tension within the normal range.

On September 19th, he returned with the complaint that his vision was hazy and his eyes painful for the past week. This came on following a cold which he acquired by sleeping in front of an electric fan.

Examination showed a bilateral plastic iridocyclitis of moderate degree. Vision was reduced in both eyes; in the right eye to 20/200; in the left to 20/100.

He was hospitalized and the treatment consisted of atropine and cortisone locally and systemically.

An immediate improvement occurred and, after a week, he returned home. The medication was continued except for a reduction in the systemic cortisone. As the inflammatory signs subsided and the vision improved, the medication was gradually lessened.

At present, he is receiving 2.5-percent cortone locally two times a day. The vision in the right eye is 20/60; left eye, 20/30 (with correction). Except for some deposits on the

posterior surface of the cornea the condition is quiescent.

The case emphasizes the hazard of the iridencleisis operation and the value of the early use of cortisone in controlling inflammation.

Glaucoma operations would seem to offer a favorable field for the development of sympathetic inflammation. For most of the operations which have been devised to relieve intraocular pressure permanently expose the uvea to infection, while the inclusion operations offer the added hazard of iris incarceration.

Although the deliberate incarceration of the iris seems contrary to good surgical procedure, iris-inclusion operations have become increasingly popular in recent years because of their efficacy in reducing intraocular pressure. Surprisingly enough, it does not appear to be a frequent cause of sympathetic ophthalmia if one can be guided by the cases reported. However, basing an opinion on conversations with various surgeons, it is more frequent than the literature indicates. Some ophthalmologists have abandoned the operation because of this danger.

Its substitution by trephination lessens somewhat, but does not eliminate, the hazard; and the trephining operation offers the added possibility of late infection and subsequent loss of the globe.

It has been frequently stated that sympathetic disease does not often follow iridectomy, for generally the operative trauma to the ocular tissues is not marked, iris prolapse is infrequent, and a keratome incision heals promptly and securely. However, cases following iridectomy have been reported.

Cataract extraction is the most common predisposing cause of postoperative sympathetic ophthalmia. Extracapsular extraction offers more danger than if the lens is removed in its capsule. Another important predisposing factor is the development of phacoanaphylaxis caused by retained lens cortex in the anterior chamber. This is probably the most frequent cause of sympathetic ophthalmia after discission of congenital cataracts. As matters stand today, the causative factor of sympathetic ophthalmia is controversial. However, from the evidence presented it would appear that infection must be present to cause sympathetic ophthalmia and that a virus is the most likely offending organism. Also, it seems probable that allergy to uveal pigment is a factor, and possibly important, in the production of the disease.

Experience indicates that the incubation period extends from one to three months, with the greatest incidence occurring during the second month. There is usually little to fear after three months have passed, and practically no danger after one year.

We have evidence to show that sympathetic ophthalmia can always be prevented if the injured eye is removed before the appearance of such changes in the fellow eye. The shorter the interval between injury and enucleation, the greater the prophylactic value. It is quite generally agreed that enucleation of the exciting eye has little or no favorable effect after sympathetic uveitis is once established.

Therapeutic procedures are, as yet, uncertain. Other than early enucleation of the exciting eye, no treatment so far suggested has been universally accepted. It is generally believed that a blind exciting eye or one with grossly faulty projection should be promptly removed, at least in early cases. On the other hand, if there is any hope whatever of retaining or regaining any degree of vision, the eye must not be removed for in the end it may be the better eye.

In cortisone and ACTH we have more powerful agents for the control of the condition than have been available heretofore. The greatest benefit from the hormone treatment will be obtained when it is given in the early stages of the process. It is yet too early to assess the true merit of cortisone and ACTH in the prophylaxis of sympathetic inflammation. They are very valuable in the control of

allergic reactions in the eye but have no effect on any underlying disease which may be the basic condition. However, the reaction is not consistent, and their use may mask the true condition of the injured eye, causing unnecessary delay in its removal.

*Discussion.* Dr. M. McCannel said that he will not diminish the number of iris inclusion procedures because of the expected fear of sympathetic ophthalmia. The precipitating cause of sympathetic ophthalmia is still too obscure and to his mind not linked closely enough with an iridencleisis procedure to encourage him to substitute another procedure.

Recently he had a case of sympathetic ophthalmia following a cataract operation. In this only a small knuckle of the iris herniated, leaving the oval pupil still intact and visible in the anterior chamber. After this minor repair, the other eye became smoky three weeks later. At the present time both eyes are quiet following the use of cortisone and other measures. The patient still has good vision. However, in this case there was no ciliary-body manipulation and only minimal iris surgery.

Dr. J. Garten said that the use of cortisone or ACTH in the treatment of sympathetic ophthalmitis is a definite and positive addition to our management of this unpleasant and tragic complication. He does not feel that we should completely forget and discard the old established prophylactic use of foreign-protein therapy. It seems to him that from a medicolegal point of view, we have not done everything unless we include the use of foreign protein, particularly in cases of perforating injuries to the region of the ciliary body. In a recent case of his own, the problem of prophylactic foreign-protein injections was discussed with a group of local ophthalmologists. The consensus of the majority was that the use of foreign protein was now unnecessary. He does not entirely agree with this point of view and probably will continue to use foreign-protein therapy in the future.

Dr. W. Bushard said that he raised the following questions concerning sympathetic ophthalmia: Has the topical use of cortone completely replaced the use of intravenous typhoid in the prophylactic and definitive treatment of sympathetic ophthalmia? Is there a medico-legal implication if one fails to use typhoid intravenously?

Dr. Richard Horns said that he agrees that Dr. Fink's clinical diagnosis of sympathetic ophthalmia in this case is correct but wonders if a person can ever be 100 percent sure of a clinical diagnosis of sympathetic ophthalmia. He thinks that cortisone and ACTH offer us the best means of therapy in cases of sympathetic ophthalmia.

Dr. V. Lindberg asked if Dr. Fink had any misgivings about atropinizing the fellow (unoperated) eye in this case of glaucoma from the standpoint of possibly precipitating an acute glaucoma in an eye which might very well have a tendency in that direction.

Dr. Fink: No.

Dr. Carl Sandt said that he had a sympathetic ophthalmia case two months ago following a cataract operation. He took the eye out and the other eye quieted down following cortone.

Dr. E. Hanson said that a few years ago, Dr. A. B. Reese presented a paper before the New York Academy in which he talked about iris inclusion and advised it. It was amazing to him at that time to see the number of men who fought the idea of iris inclusion. He believes that it certainly is the most effective surgical procedure we have and he does not think we should be afraid of it. He uses an iris inclusion when indicated and has not had any trouble.

### Melanoma of ciliary body

Dr. Howard Shaw presented a 49-year-old man who complained of difficulty reading with the left eye for about two months and then had noticed some redness on the lateral side of the bulbar conjunctiva of this eye. He also had a sensation of pressure deep in the globe.

Examination revealed he had a visual acuity of 20/20 in each eye. There was episcleral vessel engorgement of the temporal bulbar conjunctiva to the lateral side of the limbus. The left pupil reacted sluggishly to light on the temporal side and the pupil seemed slightly irregular in this area and the anterior chamber angle seemed to be quite narrow on the temporal side, also.

Intraocular pressure was: R.E., 17.1 mm. Hg (Schiøtz); L.E., 10.1 mm. Hg.

With the slitlamp, there was definite crowding of the chamber angle on the temporal side and a brownish pigment was noted at the far periphery of the iris at about the 3-o'clock position. Scattered black pigment flecks also were present on the surface of the iris of the left eye but were not present in the right eye.

Field examinations showed considerable loss of field on the nasal side in both the central and peripheral field, involving only the left eye.

Gonioscopic examination revealed considerable black pigment in the chamber angle. A brownish solid mass was seen to occupy the periphery of the iris at the 3-o'clock position and occupied the chamber angle at this site. This appeared quite solid. The pupil was dilated and it was observed that there was a solid-looking, smooth, brown mass on which the ciliary processes could be observed. This extended to about the region of the ora at the 3-o'clock position.

Transpupillary illumination showed a shadow on the sclera at the site of the presumed mass. Identation with a muscle hook was difficult over this area as compared to other areas.

Although it was thought that this could possibly be a ciliary body cyst, it seemed more than likely to be a melanoma of the ciliary body. Because of the presence of a solid iridodialysis, congestion of the overlying vessels in the quadrant occupied by the new growth, a partial paralysis of the iris in the meridian in which the tumor was located, pupil irregularity in mydriasis, pigment dust scattered over the surface of the iris, a sector of anesthesia of the cornea overlying the lesion, and a resistance to probing with the muscle hook.

---

## MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

May 12, 1953

Dr. R. A. Miller, *presiding*

### Total keratoplasty

Dr. J. Wesley McKinney reported the case of Miss B. H., aged 31 years, who was first seen July 10, 1950. She had worn contact glasses on both eyes for keratoconus which was discovered at the age of 14 years. The left eye had been quite irritable for 10 years. The vision was somewhat impaired even with contact lenses. Three weeks previously, the vision in the left eye had become suddenly blurred and the eye had a swollen, whitish appearance. Soon there developed severe continuous pain. It was only temporarily relieved by medications. She was referred for corneal transplantation.

On examination the left cornea showed a protrusion which involved the entire cornea except for a narrow zone just within the limbus. The protrusion was dome-shaped and had a ground-glass whiteness. The surface of the dome was extensively eroded. Palpation with a probe showed that the cornea was quite thick. Pericorneal injection and intense photophobia were present. The right eye showed marked keratoconus with extensive scarring in the center of the cornea. Vision with her contact glass was only 20/200, partly, in the right eye. Diagnosis of keratoconus with hydrops of the left cornea was made.

A total penetrating keratoplasty was done, using a 10-mm. trephine. The entire bulging and thickened cornea was removed and replaced by clear cornea from an eye that was enucleated as a result of an old injury.

The postoperative course was uneventful for three weeks but then some edema and later opacity developed below. The patient was last seen on September 8, 1952. The vision was 20/60+2 despite the fact that there was diffuse opacity in the lower and central portions of the cornea, with some epithelial erosion.

### Recurrent congestion after cataract extraction

Dr. Philip Meriwether Lewis presented a patient with recurrent attacks of congestion and pain following complicated cataract extraction.

Mrs. F. E. C., white, aged 47 years, had bilateral round-pupil intracapsular extraction during March, 1953. A peripheral iridotomy was performed on both eyes. No complications occurred during the 11 days that she was in the hospital or during the early weeks of her convalescence at home. About six weeks after surgery, all medication was stopped and a preliminary refraction was done, normal vision being obtained in both eyes.

Seven weeks after operation she had pain in the right eye, accompanied by congestion, which was relieved by taking aspirin and lying down. When seen four days later, the eye was free from congestion and pain but there was a definite protrusion of vitreous through the pupil of the right eye. The hyaloid was unbroken and there was some iris pigment on the temporal one half of the protrusion. Under the direct beam of the slit-lamp, the pupil contracted tightly around the vitreous, mushrooming it forward; however, at no time did it reach the posterior surface of the cornea. The hyaloid face of the vitreous of the left eye was flat and did not protrude through the pupil.

Five attacks of this pain and congestion have occurred but she had never been seen during an attack. When seen between attacks, the tension had always been normal. She was given 2.5-percent neosynephrine to use when the pain started and this seemed to her to be definitely helpful.

It was felt that the cause of the trouble was an inflammatory closure of the iridotomy, with an occasional blocking of the pupil giving rise to an accumulation of aqueous in the posterior chamber and an increased intraocular pressure.

With normal vision for distance and near and with the trouble fairly easily controlled by medication the question was: Should an iridectomy be done or not? The patient was much opposed to having any further surgery, but was it safe to permit the condition to continue indefinitely?

*Discussion.* Dr. Alice Deutsch: Has gonioscopy of the chamber angle been performed?

Dr. Lewis: Gonioscopy has not been performed and I think it is a good suggestion.

Dr. Fred Wallace: Do you know why pain is present and if you were to perform iridotomy, what procedure would you use?

Dr. Lewis: I am not able to explain the pain. It might possibly be due to temporary elevation of pressure. In answer to your question about operative procedure, I would use the Chandler method, or make a small keratome incision and do a small iridectomy.

### Acute dacryoadenitis

Dr. Oscar Dahlene, Jr., resident in ophthalmology, John Gaston Hospital, presented a 55-year-old Negress who was first seen in the Eye Clinic, Out-Patient Department, on May 8, 1953, with a complaint of swelling of both upper eyelids, pain, lacrimation, and fever for about one week. She had been treated by the General Practice Clinic for three days, receiving a total of 600,000 units of penicillin intramuscularly, and aureomycin eye ointment without improvement in her condition.

Physical examination showed marked swelling of the periorbital tissue, chemosis of the conjunctiva, a moderate amount of mucopurulent discharge, induration and en-

largement of both lobes of the lacrimal glands bilaterally, and the typical horizontal S-shape of the upper-lid margin. Photographs were taken. Smear examination revealed only polymorphonuclear leukocytes. No other adenopathy was found.

The patient stated she had had a vaginal discharge a month ago, but it was not remarkable at the present.

She was admitted to the Eye Service of John Gaston Hospital with a diagnosis of bilateral acute dacryoadenitis and treated with terramycin (250 mg., every six hours), hot compresses (20 minutes every two hours while awake), and bacitracin eye ointment locally three times a day. By the third day, the swelling had subsided greatly, and the induration and enlargement of the glands was much less.

### Multiple imagery in a one-eyed man

Dr. William F. Murrah presented Mr. J. D. L., white, aged 51 years, and having only one eye, who was seen in the office in October, 1951. His complaint and concern were that of being annoyed and disturbed by diplopia, double and triple imagery and shadows, present for the previous three months. His left eye was his only eye, vision in his right having been lost in 1940. The right eye was enucleated in 1945.

This patient stated that his vision, even though supposedly corrected with the best possible lens correction (−4.0D. sph. ⌒ −1.0D. cyl. ax. 33°/−2.5D. sph. ⌒ −1.0D. cyl. ax. 33°) was so poor and so disturbing because of the diplopia that he was unable to read with any comfort and his distant vision incapacitated him to the degree that driving his car, recognizing people, and so forth were very difficult.

He had sought medical attention from several general physicians and eye doctors without being given any real relief or help; treatments had consisted chiefly of vitamins and systemic preparations. The patient had become aware of the fact that his family and the doctors consulted believed that he was a problem for a psychiatrist and were about to suggest he be placed under the care of one since they felt that his complaints and symptoms were all mental or psychoneurotic.

Examination revealed the following: Right eye, enucleated, with prosthesis. Left eye, normal externally with a round regular 3.5 to 4.0 mm. pupil that reacted well to light. Extraocular muscles, normal. Intraocular pressure 16 mm. Hg (Schiøtz). Ophthalmoscopic and slitlamp (after dilation with 10-percent neosynephrine) revealed a slight amount of lenticular, nuclear sclerosis. The fundus was normal except for a suggestion of early foveal absorption. Vision was: O.S., 10/200 and J1 (very close). Manifest refraction was completely unsatisfactory due to the diplopia of the test letters. Following mydriasis with 10-percent neosynephrine and with the aid of retinoscopy, the following corrections improved his visual acuity, but the diplopia continued to be present: −4.0D. sph. ⌒ −1.0D. cyl. ax. 50° = 20/100, partly; also +0.75D. sph. ⌒ −5.0D. cyl. ax. 10° = 20/50; this latter seemed to be the better. With addition of a pinhole disc to either of these corrections, the visual acuity was improved to 20/40 and the multiple imagery was eliminated. A repeat refraction the next day found that −0.5D. sph. ⌒ −5.5D. cyl. ax. 10° gave the best distance acuity, 20/30 mostly, and with the addition of a pinhole disc it gave the same acuity and eliminated the double and triple vision as before. Without any correction, the patient still obtained J1, very close.

Pilocarpine (one-percent solution) was instilled into his eye twice. After a short interval, the pupillary size was reduced to 1.5 mm. Refraction with this miotic pupil found a −6.0D. sph. gave the best acuity, 20/30, and no shadows or multiple imagery. With distance spectacle glasses (O.S., −4.0D. sph. ⌒ −1.0D. cyl. ax. 33°), he was able to obtain 20/30, partly. The patient was instructed to use pilocarpine (one-

percent solution) twice daily and to continue with his present glasses. He was dismissed, extremely pleased and happy over the clearness of his newly found monocular vision which was such an improvement over his former blurred diplopia and triple imagery.

RECURRENT NODULE IN PALPEBRAL FISSURE

DR. J. B. CROSS, resident in ophthalmology at the Memphis Eye, Ear, Nose, and Throat Hospital, presented the case of a 51-year-old Negress who first entered the clinic in March, 1952, with the complaint of something growing on her right eye. She stated that the mass, lateral to the limbus in the palpebral fissure, had been present for nine weeks. At onset, the mass appeared as a red pea-sized nodule, nonpainful but tender and remained so without noticeable growth. A diagnosis of episcleritis was made and treatment with a solution of cortone, topically, was begun.

When the patient returned a month later, in April, the mass appeared less elevated and blanched. Treatment with cortone was continued.

In May, the patient returned complaining of pain for one week. The mass was larger, approximately one cm. in diameter; a smooth dome-shape, projecting two mm. in the center. A subconjunctival injection of 0.6 cc. of cortone was made near the mass and cortone continued topically.

One week later, the mass appeared much smaller and less injected. The pain was gone but tenderness remained. Treatment was now changed to cortone ointment, twice daily, and three weeks later, the mass appeared about the same. The cortone ointment was continued.

The patient was not seen again for six weeks. This time the mass appeared as a hyperemic nodule four mm. in diameter, one mm. elevated, and four mm. from the limbus. It was again painful and tender. This time the patient was tried on a solution of Trimeton maleate (an antihistamine). A week later the mass had increased in size to 1.5 cm. by 1.0 cm., raised three mm., and blanched in the center. The antihistamine solution was continued and Pyromen (four gamma, subcutaneously) was given and repeated in five days with decreased pain and tenderness and a slight decrease in size.

The pain and tenderness returned the following week with injection and swelling. The dome of the mass became yellow. The patient was put back on cortone, pain decreased but the mass remained the same for a week. A skin test with old tuberculin and serologic studies were done, revealing a four-plus Kahn reaction. Antiluetic therapy was given and low-voltage X-ray therapy to the mass.

The mass dissolved, as did the symptoms, with only a few blood vessels remaining. There was no recurrence for one month; then over one week the mass returned completely, though nonpainful and nontender. Cortone therapy was again started with blanching and decrease in size over the following week (the last of December). The first week in January, 1953, the mass returned. In size, it was 1.5 cm. in diameter and elevated 2.0 mm., with blanching in the center but without pain and tenderness.

This case is presented for suggestion as to future handling.

Daniel F. Fisher,
*Recorder for Eye Section.*

# AMERICAN JOURNAL OF OPHTHALMOLOGY

*Published Monthly by the Ophthalmic Publishing Company*



EDITORIAL STAFF

DERRICK VAIL, *Editor-in-Chief*
700 North Michigan Avenue, Chicago 11

LAWRENCE T. POST, *Consulting Editor*
640 South Kingshighway, Saint Louis 10

BERNARD BECKER
640 South Kingshighway, Saint Louis 10

WILLIAM L. BENEDICT
100 First Avenue Building, Rochester, Minnesota

FREDERICK C. CORDES
384 Post Street, San Francisco 8

SIR STEWART DUKE-ELDER
63 Harley Street, London, W.1

EDWIN B. DUNPHY
243 Charles Street, Boston 14

F. HERBERT HAESSLER
561 North 15th Street, Milwaukee 3

PARKER HEATH
Sullivan Harbor, Maine

S. RODMAN IRVINE
9730 Wilshire Boulevard,
Beverly Hills, California

JAMES E. LEBENSOHN
4010 West Madison Street, Chicago 24

DONALD J. LYLE
601 Union Trust Building, Cincinnati 2

WILLIAM A. MANN
30 North Michigan Avenue, Chicago 2

P. ROBB MCDONALD
1930 Chestnut Street, Philadelphia 3

FRANK W. NEWELL
950 East 59th Street, Chicago 37

JOHN V. V. NICHOLLS
1414 Drummond Street, Montreal

ALGERNON B. REESE
73 East 71st Street, New York 21

PHILLIPS THYGESON
220 Meridian Road
San Jose 26, California

M. URIBE TRONCOSO
500 West End Avenue, New York 24

ALAN C. WOODS
Johns Hopkins Hospital, Baltimore 5

KATHERINE FERGUSON CHALKLEY, *Manuscript Editor*
Lake Geneva, Wisconsin

*Directors:* WILLIAM L. BENEDICT, President; FREDERICK C. CORDES, Vice-President; WILLIAM A. MANN, Secretary and Treasurer; F. HERBERT HAESSLER, DERRICK VAIL, ALAN C. WOODS.



*Address original papers,* other scientific communications including correspondence, also books for review to *Dr. Derrick Vail, 700 North Michigan Avenue, Chicago 11, Illinois;* Society Proceedings to *Mrs. Katherine F. Chalkley, Lake Geneva, Wisconsin.* Manuscripts should be *original copies,* typed in *double space,* with wide margins.

Exchange copies of medical journals should be sent to *Dr. F. Herbert Haessler, 561 North 15th Street, Milwaukee 3, Wisconsin.*

*Subscriptions,* application for single copies, notices of changes of address, and communications with reference to advertising should be addressed to the *Manager of Subscriptions and Advertising, 664 North Michigan Avenue, Chicago 11, Illinois.* Copy of advertisements must be sent to the manager by the 15th of the month preceding its appearance.

*Change of address* notice should be received not later than the 15th of the month prior to the issue for which the change is to go into effect. Both old and new addresses should be given.

*Author's proofs* should be corrected and returned within forty-eight hours to the *Manuscript Editor, Mrs. Katherine F. Chalkley, Lake Geneva, Wisconsin.* Twenty-five reprints of each article will be supplied to the author without charge. Additional reprints may be obtained from the printer, the George Banta Publishing Company, 450-458 Ahnaip Street, Menasha, Wisconsin, if ordered at the time proofs are returned. But reprints to contain colored plates must be ordered when the article is accepted.


---

## DIAMOX AND THE THERAPY OF GLAUCOMA

A dramatic fall in intraocular pressure can be induced by the systemic administration of such carbonic anhydrase inhibitors as Diamox. This provides the ophthalmologist with a new weapon in the battle against glaucoma. Diamox has been used rather extensively already and most impressive results have been recorded following its administration to over 300 patients with glaucoma. In order to delineate the ultimate role of this drug in the therapy of the glaucomas and to anticipate possible toxicity, it is wise to learn the mechanism of its action as rapidly as possible. Efforts are in progress in many laboratories to learn the site and mode of lowering of intraocular pressure induced by Diamox. Only preliminary information is available at this writing, but most of the results are suf-

ficiently consistent to warrant certain hypotheses and warnings.

One of the major effects of Diamox is on the kidneys, resulting in a marked diuresis of sodium, potassium, bicarbonate, and water. However, a decrease in intraocular pressure can be demonstrated in nephrectomized rabbits. Furthermore, in the dog or man made unresponsive to the diuretic action of Diamox by ammonium chloride acidosis or by repeated doses of Diamox, ocular hypotension still follows Diamox administration. It may be concluded, therefore, that the ocular effect of Diamox is independent of the renal action.

The rapidity of the fall in intraocular pressure following intravenous injection of sodium salt of Diamox also suggests a direct effect on the eye. Carbonic anhydrase has been demonstrated in the ciliary body of the rabbit eye and it is likely that this enzyme plays a role in the formation of the aqueous humor. The finding of Kinsey that bicarbonate is present in higher concentrations in the aqueous than in the plasma is also suggestive of a functional role of carbonic anhydrase in the secretion of the aqueous. Thus, the attractive hypothesis can be formulated that the inhibition of carbonic anhydrase by *Diamox results in a suppression of inflow of aqueous humor into the eye.* This thesis obtains confirmation from the tonographic findings. Repeated tonography on the same eye reveals that Diamox lowers intraocular pressure without significant change in facility of outflow.

The use of Diamox in the short-term treatment of glaucomatous states appears to be a reasonable form of therapy. Thus, intraocular pressure can be maintained at lowered levels in such self-limited diseases as the glaucoma associated with iritis, hyphema, and glaucomato-cyclitic crises. In acute glaucoma the lowering of intraocular pressure permits more adequate gonioscopic, ophthalmoscopic, tonographic, visual, and perimetric evaluation of the eye. This affords opportunity for more accurate diagnosis and prognosis and for the institution of the most suitable form of therapy. Furthermore, as preoperative medication in all types of glaucoma, Diamox makes possible an operation on a softer, less inflamed eye with less likelihood of complications. The surgeon is offered the opportunity to perform cataract extraction as a primary procedure in those problem eyes with chronic glaucoma and cataracts. By the repeated administration of Diamox, eyes with congenital glaucoma can be whitened and the corneas cleared, permitting goniotomy under direct visualization.

The question naturally arises as to the possibility and advisability of the chronic administration of Diamox in the treatment of the glaucomas. It has been amply demonstrated that one can maintain constantly depressed intraocular pressure in glaucomatous eyes by around-the-clock Diamox for more than 10 months, but insufficient information is available to advise this form of therapy. The effects on the lens, cornea, and other ocular tissues of continuous suppression of aqueous inflow need further investigation. The important functions of the aqueous humor of supplying nutrients and carrying away waste products suggest that caution be exercised and careful observations be made in any experimental attempts to control glaucoma with Diamox. The prolonged inhibition of the high concentration of carbonic anhydrase present in the lens itself must also be considered. Furthermore, in such chronic Diamox therapy side effects, that are relatively unimportant in short-term administration, become bothersome. These include paresthesias, anorexia, and excessive drowsiness with the possibility of agranulocytosis and dermatitis.

It is important to reiterate that short-term Diamox therapy lowers intraocular pressure without changes in the disordered outflow mechanism of glaucoma. It remains to be determined whether or not chronic inhibition of the flow of aqueous will result in an "atrophy of disuse" and further compromise of the outflow channels. It must be emphasized

that Diamox does not interfere with other forms of therapy and that the indicated conventional means of therapy to improve the facility of outflow (for example, miotics or surgery) should not be discontinued or neglected whenever carbonic anhydrase inhibition is instituted.

Bernard Becker.

## CORRESPONDENCE

### A Brigadier General

Editor,
American Journal of Ophthalmology:

It was with a great deal of interest that we read in the April American Journal of Ophthalmology, page 648, of the appointment of Dr. Harold G. Scheie of Philadelphia to the rank of Brigadier General in the Army of the United States. Dr. Scheie's many fine contributions to his country and to ophthalmology make him most deserving of this honor.

I would like to point out, however, that Dr. Scheie is not the only ophthalmologist appointed to the rank of Brigadier General since Dr. William Wilmer and Dr. de Schweinitz.

Dr. H. Leroy Goss of Seattle, Washington, received an appointment to Brigadier General in 1949 while commanding the 310th Hospital Center. Dr. Goss enlisted in the National Guard Infantry in 1908 and was commissioned in the field Artillery in 1914. In 1917, he transferred to the Medical Corps as First Lieutenant and served in the A.E.F. in World War I. As a Colonel in World War II he served as commanding officer of several hospitals in Africa and the European Theater.

Between the World Wars and following World War II Dr. Goss has maintained a distinguished ophthalmic practice in Seattle and is serving as an officer in several ophthalmological societies.

(Signed) Wood Lyda, M.D.
Seattle, Washington.

## BOOK REVIEWS

The Year Book of the Eye, Ear, Nose and Throat (Series 1953-1954). Edited by Derrick Vail, M.D., and John R. Lindsay, M.D. Chicago, The Year Book Publishers, 1954. 455 pages, 126 illustrations, author and subject indices. Price: $6.00.

The abstracts covered in the 1953-1954 *Year Book* are based on publications received in the previous scholastic year (October 1952-September 1953). As usual the ophthalmic section comprises over half the book —252 pages. The effort is made to present adequately the most significant contributions in the justified belief that more attention to fewer articles is better than less attention to much more.

Included in the selections are the role of oxygen in the retinopathy of prematurity, tonography in the diagnosis of wide-angle glaucoma, lamellar scleral resection, the dangers of beta irradiation, topical cortisone in interstitial keratitis, Versene for lime burns and calcific corneal opacities, and treatment of ocular tuberculosis.

Isoniazid, though apparently bactericidal in rabbits, is only bacteriostatic in mice and men and must be combined wtih streptomycin and PAS for effective therapy. Vail, in a personal article, presents his technique of intracapsular cataract extraction in which he stresses that the cornea be tucked under the tumbling lens until final delivery. Irvine discloses that intracapsular extractions are followed not too infrequently by vitreous changes and macular degeneration.

The editorial commentary adds much to the value of the book. Vail would explain the paradox that pulsating exophthalmos occurs in congenital absence of the orbital roof but not after the Naffziger operation by the firm connective tissue barrier formed in postoperative healing. Though he agrees that topical cortisone affects adversely the acute stage of virus keratitis, he feels that it may be used later safely to lessen the density of resultant scars. Vail holds that in low-tension

glaucoma the underlying cause is vascular and that antiglaucomatous measures are useless. He endorses from experience the plastic splint in corneal transplantation as an aid in producing a flatter graft and less subsequent astigmatism. As an antidote to curare he suggests Neostigmine as more effective than physostigmine. In central angiospastic retinopathy, Vail is inclined to look for an organic basis. This view receives support from Karpe and Wising (1948) who noted such changes as an initial finding occasionally in infectious mononucleosis.

The *Year Book* can be recommended as a trustworthy aid for viewing the constantly increasing output of ophthalmic literature in proper perspective.

James E. Lebensohn.

---

TEXTBOOK OF OPHTHALMOLOGY. By M. Amsler, A. Brueckner, A. Franceschetti, H. Goldmann, E. B. Streiff, in collaboration with J. Babel, A. Bangerter, V. Bischler, J. D. Blum, R. Brueckner, E. Buerki, A. Huber, D. Klein, M. Luedin, W. Lutz, H. Suellmann, H. Weve, G. Wolf-Heidegger. Basel, S. Karger. 1954 (second edition) 927 pages, 369 illustrations, some in color, five tables, index. Price: Swiss francs 88.40.

This *Textbook of Ophthalmology* is the first of its kind to be published in Switzerland. It was planned as a substitute for the older, well-known German textbooks out of print since the last World War. The list of illustrious contributors alone suggests that the book is considerably more than a mere stop-gap but may well become one of the standards in its field.

There are three divisions: A general division, a special division, and a division on eye symptoms as related to systemic diseases. The volume has all the advantages and some of the disadvantages that result from the co-operation of several authors—brilliant as they may be individually. Some chapters are outstanding; not a single one is less than adequate.

The general division, including treatises on methods of examination, general symptomatology, and treatment, aims to introduce the student to a new and strange world. Without previous extensive instruction in the clinic and the classroom, he will not derive the full benefit of a general discussion on some very specific entities with which he is unfamiliar. Only with such a foundation will he be prepared to understand conditions mentioned merely by name but explained in later chapters.

Weve discusses the methods of examination. Without undue stress of the facts that belong to the exclusive realm of the ophthalmologist, the student can learn to perform procedures with which the general practitioner should be familiar. At the same time, he will gain enough insight to understand the theories and some of the aims of the more involved techniques of our specialty.

Amsler and Huber deal with general symptomatology. This is one of the outstanding chapters. The authors reason that the patient consults the doctor for four possible symptoms, namely a "red eye," pain, visual disturbances, or something he has noticed while looking into a mirror. They analyze the entire subject based on these facts in such a lucid fashion that the general practitioner will be able either to make a correct diagnosis or at least have a very specific idea concerning the nature and seat of the pathologic process.

The chapter on heredity by Franceschetti and Klein is exceptionally well written. It is one of the topics in the book that should be of special interest to the ophthalmologist. There is a very detailed basic presentation of the laws governing heredity in general and their implications with regard to the eye.

Several authors collaborate in describing therapy. Medical therapy is covered by Luedin and R. Brueckner. It seems almost impossible to give an outline for the ever-changing methods of treatment that will not be dated by the time of publication. R.

Brueckner deserves credit for his refreshing approach. He evaluates antibiotics in relation to other time-honored remedies. Some of these have fallen in disrepute in this country or are not used any more. Perhaps we had better examine our own enthusiastic attitude of being carried away by every innovation in the field of therapy. As is to be expected, Weve outlines only the principal of major surgical procedures without technical details. Perhaps that is why he omits mention of sutures in the description of cataract surgery.

The special division contains a very detailed and well-written chapter by Wolf-Heidegger on the anatomy of the eye and its adnexa as well as the visual pathways. The part dealing with the development of the eye is brief and to the point. Physiology by Suellmann, R. Brueckner, and A. Brueckner is not quite up to the standard of other sections; one exception is the chapter on refraction that surpasses even the masterly fashion of A. Brueckner employed in a prewar textbook (in co-operation with Meisner) on the same subject. It really is a lucid introduction to a field which is usually difficult for the beginner.

Two chapters should be singled out as truly outstanding within the framework of a single textbook on ophthalmology, namely those on glaucoma and disturbances of the extraocular muscles. Goldmann admirably covers the former. By avoiding all controversial factors, he gives an ideal introduction to this fascinating problem.

Franceschetti and Blum treat disturbances of motility in a scholarly manner. After making some introductory remarks on the anatomy and physiology of the extraocular muscles, they discuss phorias, concomitant and paralytic strabismus, and the supranuclear lesions in a manner comparable to such classics as the corresponding parts in Fuchs' textbook and Bielschowsky's chapter in Axenfeld's work. Interestingly enough, the overaction of the inferior oblique is still considered the result of a paresis of the homolateral superior oblique rather than that of the superior rectus of the other eye.

Streiff contributes the last division which deals with eye symptoms in systemic diseases. The idea of adding such a division is praiseworthy but is not carried out very successfully. Most of the salient points are stated elsewhere in greater detail. In order to systematize, he enumerates a great number of diseases with symptoms that are either quite unspecific or self-evident.

Though it is not customary to have an extensive bibliography in a textbook of this sort, brief references in some instances might enhance the value of the book. As an example, a chance remark condemning retrobulbar alcohol injections because of a possible sympathetic ophthalmia immediately arouses the interest of the ophthalmologist. Yet no further explanation is given.

The illustrations, with few exceptions, are originals. Their reproduction, as well as the splendid and tasteful arrangement of this truly first-rate contribution to ophthalmic literature is a credit to the publisher.

Stefan Van Wien.

# ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

## CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

---

## 1

### ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Baud, C. A., and Balavoine, C. **The ultrastructure of Descemet's membrane and its pathologic derivatives.** Ophthalmologica **126**:290-294, Nov., 1953.

Under the polarization microscope, normal Descemet's membrane and the hyaline network forming in the anterior chamber after severe keratitis, show the phenomenon of double refraction. (1 figure, 11 references)

Peter C. Kronfeld.

Corddry, Ellen G. **Vascular patterns in the human retina.** Bull. Johns Hopkins Hosp. **94**:148-157, March, 1954.

The character and architecture of selected vascular patterns, as demonstrated in their entirety by the periodic acid method in an unsectioned, flat preparation of the whole thickness of the normal human retina, are illustrated in a series of camera lucida drawings. (Author's summary) (11 figures, 21 references)

F. H. Haessler.

François, J., Rabaey, M., and van der Meerssche, G. **The ultramicroscopic structure of the ocular tissues. I. The retinal pigment epithelium.** Ophthalmologica **126**:347-354, Dec., 1953.

This study concerns itself with the structure of the pigment granules of the retinal pigment epithelium as revealed by the electron microscope. The granules were isolated by suspending the cells of the pigment layer (of man or cattle) in distilled water. Magnifications up to 70,000 times were employed. Under these conditions the individual granules varied somewhat in shape and size, but most of them were oblong or spindle-shaped with rounded-off extremities. The body of the individual granule showed no structural details; its surface was somewhat rough and scaly. No filamentous processes could be seen. The granules seemed to fit themselves so snugly around the outer end of the rods that any movements of the pigment granules (under the influence of light) seemed highly unlikely. The authors' findings are strikingly different from those of Sebruyn (Am. J. Ophth. 33: 1621, 1950). (18 figures, 4 references)

Peter C. Kronfeld.

Friedman, Benjamin. **Nature of the corneal "canals" produced by perilimbal in-**

**jections of hydrogen peroxide.** A.M.A. Arch. Ophth. **50**:688-695, Dec., 1953.

Freshly enucleated steer eyes were injected with hydrogen peroxide in an attempt to verify the results of Magnus and Stübel. From these studies the author concludes that the superficial and dry corneal canals of Krassius, Magnus and Stübel are artefacts. However, there is some evidence by entoptic methods, that some kind of channels do exist. (7 figures, 6 references) G. S. Tyner.

Prince, J. H. **Vertebrate visual centres.** Brit. J. Physiol. Optics **11**:73-87, April, 1954.

This paper is complete with 15 illustrations showing the anatomy and physiology of the central nervous system of various animals. Functional variations due to differences in crossing fibers at the chiasma were discussed at length. Especially interesting was the discussion of the function of the lateral geniculate bodies in the primates, compared to lower animals. Paul W. Miles.

## 2

## GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Bruna, F. **Experimental intraocular infection of the rabbit with vaccinia virus.** Boll. d'ocul. **32**:723-740, Dec., 1953.

The virus, a suspension of chorioallantoic membranes of chick embryos infected with vaccinic lymph, introduced into the anterior chamber or into the vitreous body, caused severe inflammation of the anterior uvea, and corneal infiltration throughout the parenchyma. This inflammation was followed by severe atrophy of iris and ciliary body. The cornea became ectatic. Very small quantities of the virus sufficed to evoke this reaction. The infection of the anterior chamber could be transmitted in serial experiments from animal to animal. Active material from iris and aqueous humor could be recovered by egg culture long after the inoculation. Retina and optic nerve were not susceptible to the infection.

When the infectivity was tested at various times after inoculation, true proliferative activity of the elementary bodies was found. The inner eye proved to be more susceptible to the virus than cornea or skin. Resistance to revaccination occurred 25 days or less after inoculation into the anterior chamber. (3 figures, 25 references) K. W. Ascher.

Halbert, S. P., Swick, L., Sonn, C., and Locatcher-Khorazo, D. **Ocular antibiotic-producing bacteria in normal eyes and in conjunctivitis.** A.M.A. Arch. Ophth. **51**:7-14, Jan., 1954.

This report summarizes the survey findings relating to antibiotic-producing strains of bacteria in normal eyes and eyes with conjunctivitis. The subjects were inmates of a feebleminded institute. No significant difference was found in the incidence of this type of bacteria in the normal and inflamed eyes. No correlation was found between the prevalence of antibiotic producing strains and the incidence of conjunctivitis. The same bacterial flora were found in both normal eyes and eyes with conjunctivitis. (7 tables, 23 references) G. S. Tyner.

Lemoine, Albert N., Sr. **Common manifestations of allergy in ophthalmology.** Tr. Am. Acad. Ophth. **58**:125-127, Jan.-Feb., 1954.

The author mentions the most common symptoms of allergy of the lids, conjunctiva, cornea, uveal tract and lens. He states that retinitis and optic neuritis may be due to an allergy from foci of infection, and to tuberculoprotein. (7 references) Theodore M. Shapira.

Prewitt, Leland H. **Psychogenic allergy as it pertains to retrobulbar neuritis and**

**otolaryngology.** Tr. Am. Acad. Ophth. **58**: 111-115, Jan.-Feb., 1954.

Psychosomatic factors, often with allergic reactions, could possibly be the causative agent in many cases of retrobulbar neuritis. (6 references)

Theodore M. Shapira.

## 3

## VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Allen, R. A., and Friedenwald, J. S. **Distribution of substrate-specific alkaline phosphatases in the ocular tissues.** A.M.A. Arch. Ophth. **50**:671-684, Dec., 1953.

Studies were done on fresh frozen eyes of albino rabbits to localize in the ocular tissues certain substrate specific enzymes which hydrolyze various phosphate esters in an alkaline environment. The authors state that the variety of enzyme composition in different cells is an expression of their varied metabolic activities and of the metabolic interactions which contribute to organ functions. Two enzyme systems, A and B, were studied. The A system, which is active in the absence of magnesium, is substrate specific, while the B system, which requires activation by magnesium, attacks all four substrates used. It was concluded that the B enzyme was not a necessary component of the glycolytic system or that the inactive tissues contain an endogenous inhibitor. The A enzymes, which are important in transport of glucose in renal and intestinal tissue, were not found in the ocular tissues. This is evidence against the active transport of glucose into aqueous fluid. (36 figures, 1 table, 6 references)

G. S. Tyner.

Bottino, Carlo. **The effect of tonometric measurements of the speed of the aqueous elimination.** Ann. di ottal. e clin. ocul. **79**:33-40, Jan., 1953.

The speed of aqueous elimination was measured by estimating the fall of the ocular tension under continuous application of the tonometer and under the effect of various agents. Pilocarpine and priscoline, which are vasodilators, cause an increase, adrenaline, privine and "simpamina," which are vasoconstrictors, retard the flow of aqueous out of the eye. (5 references)

John J. Stern.

Caselli, P., and Schumacher, H. **The micro-electrophoretic determination of lysozyme in tears.** Klin. Monatsbl. f. Augenh. **124**:148-154, 1954.

This determination is more reliable than the biological methods. Lysozyme is an alkaline protein with an isoelectric point near pH 11. In a neutral solution it will move toward the cathode. Lysozyme could be found in tears, saliva, and egg white. (4 figures, 31 references)

Frederick C. Blodi.

Ennema, M. C., and Zeeman, W. P. **Venous occlusions in the retina.** Ophthalmologica **126**:329-347, Dec., 1953.

The very extensive, clinical and pathohistologic material on venous retinal occlusion of Zeeman's clinic in Amsterdam is reviewed. The findings and interpretations are in close agreement with those of previous investigators of the subject (Verhoeff, Coats, Scheerer and Foster Moore. Since actual thrombotic processes are rare, venous occlusion is the preferable term. The common occurrence of branch occlusions distally to an abnormal arteriovenous crossing makes it highly probable that the immediate cause of the occlusion is mechanical. Comparable abnormal (mechanical) relationships may exist between artery and vein in the lamina cribrosa. Trunk occlusion has the characteristics of a simple persistence of the physiologic collapse of the vein. This failure of the vein to reopen may be due to a reduced flow volume which, in turn may be caused by arteriosclerosis or circulatory embarrassment by a coexistent

glaucoma. In a few cases a functional vascular disease may play a part. (12 figures) Peter C. Kronfeld.

Gemolotto, G. **Soluble proteins in the lens of rabbits with retino-choroiditis, experimentally produced with sodium iodate.** Gior. ital. oftal. **4**:509-516, Sept.-Oct., 1953.

In the course of work to determine the effect of retinal pigmentary degeneration on the other ocular tissues, the author, using the method of electrophoresis, has assayed the concentration of soluble proteins in the lens of rabbits whose retina had become degenerated as a result of the intravenous injections of sodium iodate. He found that there was a variation of the relative proportions of alfa and beta globulins in the lens. He also found an almost constant diminution of the albumin-globulin quotient in the serum. He suggests that the variation in the lens globulins, which is not unlike that found in cataract, may be due to the fundus changes. V. Tabone.

Hummelt, Klaus. **The blood-aqueous barrier after intramuscular injection of radioactive penicillin in rabbits.** Klin. Monatsbl. f. Augenh. **124**:154-158, 1954.

The primary and secondary aqueous were examined and their radioactivity determined after the injection of one half to one million units of penicillin with a radioactive isotope of sulphur. The penicillin concentration in the primary aqueous of a normal eye is minimal, but it increases markedly in the secondary aqueous or in an inflamed eye. The prophylactic and therapeutic use of intramuscular penicillin is therefore indicated. (3 figures, 8 references) Frederick C. Blodi.

Ishihara, F. **Biochemistry of cataract.** Acta Soc. Ophth. Japan **57**:1495-1500, Dec., 1953, and **58**:96, Jan., 1954.

Vitamin C prevents the development of naphthalin cataract. Beta-naphthoquinone is excreted in the urine of naphthalin-treated rabbits. It is also a cataract-developing substance. The development of cataract by beta-naphthoquinone can be prevented by 5-oxy-anthranilic acid. (3 figures, 8 tables, 8 references) Yukihiko Mitsui.

Juzwa, Josefa. **Effect of the hydrazide of isonicotinic acid in some eye diseases.** Klinika Oczna **23**:267-272, 1953.

Hydrazide of isonicotinic acid was used on 20 patients with eye diseases. The results were encouraging despite the fact that the tuberculous etiology of an eye disease is hard to establish. All of the cases were chosen because of poor response to treatment with streptomycin and PAS. Definite improvement was seen in 30 percent of cases, moderate improvement in 40 percent, little improvement in 20 percent, one case was without change and one became worse. In most cases there was no recurrence. Six cases are presented in detail to illustrate the results of treatment with hydrazide. (1 table) S. Brandon.

Kishimoto, M. **Anterior pituitary hormone and vitamin $B_1$ distribution in aqueous and vitreous.** Acta Soc. Ophth. Japan **57**:1481-1486, Dec., 1953.

When 200 RE of the anterior pituitary hormone was given in the rabbit, there was a slight increase in vitamin $B_1$ content in the aqueous and vitreous. When the hormone was given in combination with vitamin $B_1$, however, it did not result in a greater increase in the vitamin in the organs mentioned above, nor in the blood and spinal fluid, than when the vitamin was given alone. (2 tables, 16 references) Yukihiko Mitsui.

Kubota, K., and Morikawa, S. **Influence of ACTH on retinal metabolism.** Acta Soc. Ophth. Japan **58**:128-130. Feb., 1954.

When 4.0 mg. ACTH was injected into rabbits daily for three days, the carbohydrate breakdown and the aerobic glycolysis decreased 15.3 percent and 12.7 percent respectively as measured by Warburg's method. (3 tables, 15 references)

Yukihiko Mitsui.

Lijo Pavia, J. **The eyeground in experimental hypercholesterolemia.** Arch. oftal. Buenos Aires **28**:546-558, Dec., 1953.

Certain disturbances in the lipoid metabolism may give rise to changes similar to arteriosclerotic changes. Five dogs received a cholesterol-rich diet for four months. In all of them arteriolar constriction and crystalline deposits in the retina were observed; this last sign was reversible. Two of the dogs also had hyperemia of the disc. (4 figures, 20 references)

Walter Mayer.

Miglior, M., and Pirodda, A., **Lysozime in the cornea of man, and some domestic animals.** Gior. ital. oftal. **4**:494-497, Sept.-Oct., 1953.

This work is part of a study to determine the relation between lysozime and natural immunity to herpetic infection. Experiments were carried out on man, horse, ass, dog, rabbit and guinea pig. It was determined that the human cornea, as well as the cornea of some domestic animals, exhibited lysozimic activity, and that there was evident a relation between this activity and immunity to herpetic infection. In the animals used there was no relation between the lysozime concentration and the position of the animal in the zoological ladder.

V. Tabone.

Nagata, N., Kurimoto, S., and Matsuka, M. **A study of consensual ophthalmotonic reaction.** Acta Soc. Ophth. Japan **58**:38-54, Jan., 1954.

A puncture of one eye of a rabbit resulted in a fall of ocular tension in the second eye, but the changes in the tension were not parallel in both eyes. In one of the cases a prolapse of the iris resulted from the puncture and an increase in tension followed in the first eye; nevertheless, a decrease in tension resulted in the second eye.

When the autonomic nerve was blocked by tetraethylammonium, the consensual reaction was impeded. Narcosis by evipan-sodium impeded the reaction while urethane did not. The authors consider the consensual reaction to be a reflex through the autonomic nerve and regulation center which is supposed to be in the midbrain. (2 figures, 9 tables, 44 references)

Yukihiko Mitsui.

Ohara, H. **Effect of tetraethyl-ammonium on the ocular tension.** Acta Soc. Ophth. Japan **58**:239-247, & 259-268, March, 1954.

When tetraethyl-ammonium is injected into the rabbit intravenously there is immediately a considerable drop of blood pressure with a slight drop of ocular tension. There is also a considerable interference with the regulation of tension of the eye, and this is easily demonstrated by a compression test. In the same rabbit there is an acceleration of fluorescein permeability through the blood-aqueous barrier. When the drug is given subconjunctivally there is a considerable drop of ocular tension, with mydriasis, in half an hour. There is also an acceleration of fluorescein permeability as well as a decrease of protein content in the aqueous. (3 figures, 6 tables, 41 references)

Yukihiko Mitsui.

Purcell, E. F., Lerner, L. H., and Kinsey, V. E. **Ascorbic acid in aqueous humor and serum of patients with and without cataract.** A.M.A. Arch. Ophth. **51**:1-6, Jan., 1954.

Ascorbic acid exists in higher concentration in the aqueous than in the blood. It has been stated that this difference in

concentration is somehow attributable to the presence of the crystalline lens. The authors found that the concentration of ascorbic acid in the aqueous varied approximately as the concentration in serum regardless of whether the lens was cataractous or the eye aphakic. Samples of aqueous were withdrawn from human eyes with normal and cataractous lenses and from two aphakic eyes. The authors believe from these studies that it is improbable that the higher concentration of ascorbic acid in the aqueous as compared to that in serum can be attributed to synthesis by the lens. They favor the hypothesis that ascorbic acid enters the aqueous from the ciliary body. (14 references)

G. S. Tyner.

Puxkandl, H., Ibrahim, M. G., and Tewfik, H. **The diffusion of isonicotinic acid hydrazide into the aqueous.** Ophthalmologica **126**:387-393, Dec., 1953.

The rate of entry of isonicotinic acid hydrazide into the aqueous of the rabbit was determined after intravenous doses of 10 mg./Kg. of body weight and after subconjunctival doses of 12.5 mg. of the drug. In both instances demonstrable and therapeutically effective concentrations of the drug were shown to prevail in the aqueous for several hours after the injection. (1 figure, 2 tables, 9 references)

Peter C. Kronfeld.

Redi, Francesco. **Biochemical modification of preserved corneal tissue.** Rassegna ital. d'ottal. **22**:556-565, Nov.-Dec., 1953.

In two previous articles it has been shown that, when preserved at low temperatures, cornea, as a part of the globe, shows a progressive increase in weight, while excised cornea loses weight. The imbibition of fluid is probably due to reduction of the impermeability of the posterior corneal surface. The mucopolysaccharides show their point of maximum insolubility at pH 4 in either type. There is little variation in the extractibility of the mucoids whether the corneal tissue is excised or not. (8 figures, 2 references)

Eugene M. Blake.

Reis, J. L. **Histochemical localization of alkaline phosphatase in the retina.** Brit. J. Ophth. **38**:35-38, Jan., 1954.

Previous attempts to demonstrate alkaline phosphatase in the retina have been unsatisfactory in that the end product was a black, insoluble, cobalt sulphide, which was indistinguishable from normally present melanin pigment. The author describes a technique for this determination using alpha-naphthol phosphate in the presence of a diazonium-compound. The end product is red and is easily distinguished from melanin. (2 figures, 9 references)

Morris Kaplan.

Rissoli, Emilio. **A new surface anesthetic—novesina.** Rassegna ital. d'ottal. **22**:583-586, Nov.-Dec., 1953.

Novesina is the chlorhydrate of butylaminobenzoil-diethylaminoethanol. It has been tested in 0.2 percent solution for surface anesthesia in normal and diseased eyes. Its advantages are rapid action, perfect tolerance, lack of burning sensation or disturbance of corneal epithelium. At times a transitory dilatation of conjunctival vessels is noted after several applications, but no true allergy is induced. There is no effect upon the size of the pupil. Two seconds after the application of two drops of novesina a foreign body may be removed without pain, the anesthesia lasting for 10 to 12 minutes. The drug is thermostabile.

Eugene M. Blake.

von Sallmann, L., Monox, C. M., and Drungis, A. **Effects of beta irradiation on the rabbit lens.** A.M.A. Arch. Ophth. **50**: 727-736, Dec., 1953.

Because of the increased use of B ir-

radiation by ophthalmologists in a wide variety of ocular lesions, experimental studies were made to determine the effects of these rays on the rabbit lens. The $Sr^{90}$ applicator was used on mature chinchilla rabbits. One eye was irradiated and the other used as a control. The applicator was applied to the upper limbus and surface doses of 2,000; 5,000; 10,000; and 20,000 rep were given. Eyes were removed for study 1, 4, 7, 14 and 28 days after irradiation. Another group of rabbit eyes were exposed to X-ray doses of 100, 500 and 1500 r.

The $Sr^{90}$ treated animals showed damage to the nuclei of the germinative zone of the lens epithelium closest to the irradiated limbus. In previous experiments these changes ultimately resulted in cataractous changes. The X-ray treated animals showed comparable lens changes. (5 figures, 9 references) G. S. Tyner.

Sampaolesi, Roberto. **Laboratory experiences with fluoresceine**. Arch. oftal. Buenos Aires **28**:559-564, Dec., 1953.

In order to know the status of the blood—aqueous barrier, the author injected fluoresceine intravenously and then determined the fluoresceine concentration in the blood and aqueous. This was done first in 500 normal individuals, and later, in a second stage, the variations in the concentration were studied in patients with different ocular diseases. The concentration of fluoresceine in the aqueous 30 minutes after intravenous injection is 100 times less than the concentration in the blood. The author concludes that the concentration of fluoresceine is not affected by body weight, as long as the intravenous dose is the same. The test should be valuable in the early diagnosis of many ocular diseases, in the selection of the type of surgery to be performed in glaucomatous patients, and in the decision to enucleate an eye before symptoms of sympathetic ophthalmia appear. (3 figures, 2 tables) Walter Mayer.

Sapuppo, C. **Influence of B-piridilcarbinol on the blood-aqueous barrier.** Gior. ital. oftal. **4**:503-508, Sept.-Oct., 1953.

After reviewing some of the pharmacological effects of nicotinic acid and B-piridilcarbinol, the author reports on work carried out on 20 persons, to find out the effect of B-piridilcarbinol on the blood-aqueous barrier, compared to that of nicotinic acid. He discovered that in all the experiments B-piridilcarbinol increased the permeability of the blood-aqueous barrier more rapidly and effectively than nicotinic acid. He agrees with the opinion expressed by others that this drug in addition to its vasodilator action stimulates the para-sympathetic system.

V. Tabone.

Sbordone, Girolamo. **The chloride concentration of the aqueous and transparency of the lens.** Ann. di ottal. e clin. ocul. **79**:55-63, Jan., 1953.

Sodium chloride injections of ½ cc. of various concentrations were administered subconjunctivally to one eye of rabbits. The chloride content of the aqueous could be shown to be increased a short time after the injection of 5 and 10 percent concentrations, and the optic density of the lens was augmented. The sodium chloride content of the lens was unaltered in all cases, even where the aqueous concentration was elevated. (32 references)

John J. Stern.

Simonelli, M., and Rizzini, V. **Isonicotinic hydrazyde in ophthalmology.** Gior. ital. oftal. **4**:405-408, Sept.-Oct., 1953.

In view of the greater concentration of the drug in the anterior chamber after subconjunctival injection, an attempt was made to determine whether a greater protection to ocular tuberculosis could be obtained by this method. Tests were carried out on 16 rabbits which were artificially inoculated; four received no treatment and served as controls. The other 12 were divided into two lots, one receiving treat-

ment on the second day after inoculation and the other on the fifteenth day. The treatment was the daily injection of 10 mg. of the drug subconjunctivally in the left eye only for a period of 40 days. A further period of 60 days followed during which the animals were kept under observation. Then they were killed and the eyes examined.

The controls showed evidence of violent inflammation and after the three-month period they showed no evidence of spontaneous healing. Treated animals showed a great attenuation of inflammatory signs, and these were again much less marked in the eyes which had had subconjunctival injections. Animals treated on the second day after inoculation showed the least reaction. It would appear that subconjunctival injection of the drug exerts a powerful protective action in ocular tuberculosis. V. Tabone.

Soya, T. **Vitamin C metabolism of the lens.** Acta Soc. Ophth. Japan **57**:1503-1505,-Dec., 1953.

Naphthalin impedes the synthesis of vitamin C from di-keto-gulonic acid in the crystalline lens. The manganese ion accelerates the synthesis while the calcium ion impedes it. (5 tables, 12 references) Yukihiko Mitsui.

Yamashita, K. **Colimycin® (Colistin-Lion) in ophthalmology.** Acta Soc. Ophth. Japan **58**:35-37, Jan., 1954.

Colimycin is a new antibiotic found in Japan. It has much the same spectrum as polymyxin. It is extremely effective against Koch-Weeks as well as pyocyaneus infection of the eye. A 0.1 percent ointment applied four times daily effected a cure in three cases of typical Koch-Weeks' infection in a few days. Subcutaneous or intraocular infection with pyocyaneus was treated with local injection with benefit. (5 references) Yukihiko Mitsui.

## 4

## PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Alajmo, A. **Research and measurement of aniseikonia with the space eikonometer.** Gior. ital. oftal. **4**:436-443, Sept.-Oct., 1953.

Aniseikonia was measured with a space eikonometer in a patient with anisometropia while wearing ordinary glasses and contact lenses. The author confirmed the belief that contact lenses considerably reduce this condition. W. Tabone.

Charnwood, Lord. **Fusion in binocular vision.** Brit. J. Physiol. Optics **11**:65-72, April, 1954.

Charnwood rejects the theories of binocular vision in which one of each pair of corresponding retinal areas inhibits the other (Verhoeff, Asher) in favor of the more conventional view in which the two images join in consciousness. Certain experiments are described which are so interpreted. The pure projection theories in which an object is perceived in space at the intersection of the two visual lines must be revised, however, to make way for experimental facts of distance perception such as the horopter and the subjective parallel and subjective equidistant, which do not conform to Euclidean or straight line geometry. The chief experiment contradicting the Asher suppression theory is a stereo pair of a crank. Breaks in one part of the picture continue to be perceived at two depth levels simultaneously without diplopia. If suppression were the rule, one should not be aware of the defect seen only by one eye. Charnwood does not rule out suppression of one image in physiological diplopia. Such would be just as natural as ignoring background noise in the street. He relates some of Asher's experimental findings to fixation disparity and to the influence of heterophoria. Paul W. Miles.

Falkowski, Zofja. **Retinal correspondence.** Klinika Oczna **23**:231-248, 1953.

A definition of binocular vision and normal and abnormal retinal correspondence is presented. Methods of examination and measuring of abnormal retinal correspondence are described which include the after image test, the testing with an amblyoscope, and a method devised by the author. This method uses the principles used in a troposcope. Cover test, diplopia test and Maddox rod are used for determination of deviation of the eye at the subjective angle. A new test is described by the author. It consists of a 50 to 60 cm. board which is placed in front of the patient's face in a central vertical plane. Straight lines are drawn on both sides of the board at the same level. The patient looks along the lines seeing one line with the right eye and the other line with the left eye. Openings are cut in the board at different distances. A fixation object can be placed in one of them which can be seen with both eyes. The author describes in detail what happens to the relative position of the lines and the fixation object in different abnormal conditions of retinal correspondence. All three methods determine the position and the degree of abnormal correspondence. When the suppression is not too deep orthoptic exercises may be successful in restoring normal correspondence. The author discusses the phenomena of suppression. (7 figures, 1 table, 41 references)

S. Brandon.

François, J., Stefens, R., and Derouck, A. **Electroretinoencephalography in clinical ophthalmology.** Ann. d'ocul. **187**:40-69, Jan., 1954.

The electroencephalographic "syndrome," which the authors obtain by a simultaneous study of the electrical responses of the retina and visual cortex to a light stimulus, provides objective and subjective responses which make it possible to study the relationships between retinal activity and cortical activity on the one hand and between electrographic and subjective responses on the other. (26 figures, 26 references)

John C. Locke.

Hattwich, Robert G. **Dark adaptation to intermediate levels and to complete darkness.** J. Optic. Soc. America **44**:223-228, March, 1954.

Dark adaptation curves were measured carefully at various illumination levels with test stimuli, either foveal or parafoveal. The results show that the dark adaptation rate is most rapid in complete darkness. The curve of the cone adaptation shows a greater slope, while the time of the break in contour due to rod adaptation comes as much as 140 seconds sooner. Rod adaptation continues more rapidly in complete darkness. This refutes other experimental work indicating that dark adaptation is just as rapid when darkness is gradually increased. The shape of the curves confirm current theories about the photochemistry of visual purple and the corresponding substance in the cones. The curves taper off gradually, as would be expected by the laws of mass action.

Paul W. Miles.

Haycock, J. B. **Flicker perimetry.** Brit. J. Physiol. Optics **11**:94-106, April, 1954.

A five-centimeter disc target was illuminated by a rad neon stroboscope lamp for use with a one-meter tangent screen for testing visual fields in office practice. The favorable reports of Riddell, Weekers, Miles and others were confirmed. Flicker perimetry promises an office procedure which is more sensitive in the early diagnosis of certain eye and brain diseases than standard methods. It amounts to a tridimensional technique, which was illustrated in comparison to

the Traquair island. In a few minutes, a visual field could be obtained comparable to tests with targets of several sizes, by the standard method. Paul W. Miles.

Hofstetter, H. W. **Some interrelationships of age, refraction, and rate of refractive change.** Am. J. Optometry **31**:161-169, April, 1954.

This is a statistical study of 579 patients with the usual incidence of hypermetropia and myopia in all age groups. Spherical equivalents in both eyes were determined upon two occasions considerably separated in time. The rate of change of refraction was compared in various age groups. This longitudinal method of study of myopia was compared to the cross-sectional usually used. The slight changes in young people with slight hypermetropia compared to marked changes in the same age group with slight myopia were attributed to the fact that a patient with rapidly decreasing hypermetropia does not come for refraction until myopia actually occurs. The rate change in young hypermetropes therefore was considered atypical in any published series because of patient self-selection. The question whether undercorrection of myopia creates conditions allowing an increasing rate toward myopia remains unanswered. Paul W. Miles.

Hurvich, L. M., and Jameson, D. **Spectral sensitivity of the fovea. III. Heterochromatic brightness and chromatic adaptation.** J. Optic Soc. America **44**:213-222, March, 1954.

A series of experiments is reported on the foveal threshold luminosity curve, with variations due to pre-adaptation to various colors. The 1° luminosity function is shown to undergo regular and consistent changes, with changes in the color adaptation and to be further dependent on the ratio of background illumination. The main effect of adaptation to a given hue is a relative decrement in the luminosity of the spectral stimuli which evoke that hue. Paul W. Miles.

Miles, Paul W. **Visual effects of pink glasses, green windshields, and glare under night-driving conditions.** A.M.A. Arch. Ophth. **51**:15-23, Jan., 1954.

Modern green automobile windshields filter out red and infrared rays which carry heat. For night driving they are the worst color selection because almost two thirds of headlight energy is in the red end of the spectrum. One third of headlight energy only is in the range to which a green windshield is transparent. The use of pink tinted spectacles cuts down the transmission of light even more. The average luminosity through pink glasses is 71.8 percent as apposed to 100 percent through clear crown glass. Pink glasses alone reduce visual acuity from 20/20 to 20/40. Combined with a green windshield, normal visual acuity is reduced to 20/60 under normal night driving conditions. (4 charts, 13 references) G. S. Tyner.

Morgan, Alan A. **Stereoscopic vision in relation to photo-interpretation.** Brit. J. Ophth. **38**:29-34, Jan., 1954.

The stereoscopic interpretation of photographs took on additional significance during the war and it appears that it will grow in importance; this interpretation becomes more acute with the specialized knowledge of the viewer as well as with his stereoscopic sense. This report is the result of an attempt to improve or to train true stereoscopic vision. The training was carried out on Holme's stereoscopes, using the stereograms of Livingston. The group being trained was compared with a control by a group not receiving training, but interpreting the same stereo-photographs. From the studies it was concluded that the trained

personnel showed some improvement and was probably a little better than the untrained, but there actually was little difference between the two groups since the untrained also improved simply by studying the photographs. Suppression, either partial or complete, played a big role in stereoscopic sense, while refractive errors had no appreciable effect. (6 tables, 1 reference) Morris Kaplan.

Shikano, S. **Mode of light perception in the retina.** Acta Soc. Ophth. Japan **58**: 167-173, & 268-273. Feb. & March, 1954.

After a description of a series of functional experiments Shikano states that, in contrast to Polyak's experiments, Rico's law of spacial summation is valid for vision in the fovea, where, according to Polyak, one to one correspondence of visual cell with nerve fiber exists. He thus contradicts the anatomical explanation of spatial summation by Polyak. He further states that, in general, when the eye is adapted to a less intense light, a less intense illumination of an object is enough to give the same resolving power to the eye. However, the converse is true for the peripheral vision of an eye adapted to a poor light. (8 figures, 1 table)

Yukihiko Mitsui.

Sloan, L. L. **Achromatopsia congenital.** J. Optic. Soc. America **44**:117-128, Feb., 1954.

This paper describes 19 patients with congenital achromatopsia, which was complete in 16. Their visual acuity varied from 20/70 to 20/200. All showed photophobia without complaint of pain. There was blinking and poor fixation. Only four had nystagmus. Cases were proven by tests of color discrimination. The fundi were normal in four patients; the others showed slight irregularities of the macula and one had pale discs. The visual fields were normal and no central scotoma could be demonstrated. Twelve knew relatives who had defective color sense and five were children of consanguineous parents. Tests of foveal vision in these subjects showed evidence of rod, as well as cone function, which was attributed to eccentric fixation.

Paul W. Miles.

Wirth, A. **Statistical distribution of the values of the equation of Rayleigh with Nagel's anomaloscope.** Gior. ital. oftal. **4**: 423-435, Sept.-Oct., 1953.

Rayleigh's equation, was considered from the point of view of determination of threshold values. Using the method of minimal changes, the author came to the conclusion that the combined method, that is starting from the extreme left, and from the extreme right, alternately or at random, gave the more reliable results; at least five different measurements are recommended for each person. He believes that each eye must be considered as a statistical unit, and that the mean readings of each eye tested separately does not represent the actual binocular performance. The frequency distribution curve was found according to that of Gauss. Some reasons for normal variation are discussed. V. Tabone.

## 5

### DIAGNOSIS AND THERAPY

Bein, Kazimierz. **Principles of a rational structure of a biomicroscope.** Klinika Oczna **23**:293-296, 1953.

The author states the features which a good slitlamp should have. (2 figures, 7 references) S. Brandon.

Dobree, J. H., and Fincham, E. F. **Angle of the anterior chamber viewed by transmitted light.** Brit. J. Ophth. **38**:49-51, Jan., 1954.

Certain structures of the region of the anterior chamber angle are well seen in transillumination and an instrument for transillumination is described. An endoscopy lamp is attached to the rim of a

Goldman gonioscope and used in conjunction with the Zeiss slitlamp so that direct illumination can be carried out at the same time. The chamber angle is seen considerably more clearly than by ordinary methods. 1,000 patients have been successfully studied by means of this instrument. (2 figures) Morris Kaplan.

Fleischer, E. **Plastic canula for the lacrimal duct.** Klin. Monatsbl. f. Augenh. **124**:202, 1954.

Instead of the usual metal canula the author prefers a plastic one, 0.8 mm. wide and 5-8 cm. long. It is more elastic and can cause no injury. (1 figure)

Frederick C. Blodi.

Juszkowa, Krystyna. **Treatment of tuberculous diseases of the eye with streptomycin.** Klinika Oczna 23: 259-266, 1953.

The author discusses methods of treatment of tuberculosis of the eye with special emphasis on the use of streptomycin. Thirty-five patients with different tuberculous conditions of the eye were treated. Streptomycin was injected under the conjunctiva or retrobulbarly depending on the location of the disease. 50 to 100 thousand units were injected daily or every second day; up to 2 million units were used. In 12 cases of chorioretinitis fresh exudates were absorbed and pigmented scars formed. There were two recurrences. Visual acuity also improved after the treatment of chorioretinitis, central retinitis, uveitis and parenchymatous keratitis. Three cases are described for illustration. The author concludes that streptomycin can be used in all cases of ocular tuberculosis and that it shortens the time of treatment. Local use of it proved to be safe and without side reactions. (5 figures, 16 references)

S. Brandon.

Langham, M., and Wybar, K. C. **Fluorophotometric apparatus for the objective determination of fluorescence in the anterior chamber of the living eye.** Brit. J. Ophth. **38**:52-57, Jan., 1954.

Numerous attempts have been made to study with mathematical certainty the circulation of a dye within the aqueous of the living eye but they have been unsatisfactory. A fluorophotometer, which consists of a photo-electric cell attached to the Haag-Streit slitlamp, is described which permits an objective determination of the degree of fluorescence under both experimental and clinical conditions. With it one can determine differences in the permeability of the blood-aqueous barrier under varying clinical conditions. (4 figures, 6 references) Morris Kaplan.

Leopold, Irving H. **Therapeutics of ocular allergy.** Tr. Am. Acad. Ophth. **58**: 136-141, Jan.-Feb., 1954.

In all cases of ocular allergy one should identify the allergen and this should be removed from the environment, or the patient properly desensitized to its effect. When these measures are not possible, there are several palliative means to be used, such as the use of adrenalin, antihistaminics and the steroidal hormones. Every effort should be made to establish the correct diagnosis. Patients should not be referred to an allergist for the necessary tedious work-up until one is reasonably sure that he has excluded other possible diagnoses. (24 references)

Theodore M. Shapira.

Penzholz, H., and Mildbraed, I. **Ocular symptoms in intracranial aneurysms.** Klin. Monatsbl. f. Augenh. **124**:179-188, 1954.

This is a report of two cases diagnosed by arteriography and cured by an operation. The first patient was a 44-year-old man with an aneurysm of the internal carotid. The second patient, a 52-year-old woman, developed an arterio-venous fistula in the cavernous sinus after trauma.

In both cases the ligation of the internal carotid was preceded by the ligation of the common carotid on the same side. The arteriography was done percutaneously. (8 figures, 42 references)

Frederick C. Blodi.

Rosetti, D., and Santelli, F. **A new attachment to the Maggiore perimeter for the determination of critical flicker fusion frequency.** Ann. di ottal. e clin. ocul. **79**:3-10, Jan., 1953.

A compact attachment is described which can be used with the Maggiore perimeter for the determination of the flicker fusion frequency. It was tested on normal and diseased eyes and the values obtained are identical with those of other authors using more complicated apparatus. (9 figures, 9 references)

John J. Stern.

Segal, Pawel. **Water chamber with glasses.** Klinika Oczna **23**:297-299, 1953.

The author describes a water chamber attached to the spectacle frames in order to protect the eye from drying. Paralysis of the ophthalmic branch of the trigeminal nerve was the reason for wearing this protective device. The water chamber was made of plastic and attached to the glasses regularly worn by the patient. The vision in the involved eye was low enough so that the vapor on the glass did not interfere with seeing. (2 figures)

S. Brandon.

Venturi, Giorgio. **Clinical considerations concerning the finding of capillary aneurysms of the conjunctiva in cases of diabetes, hypertension, arteriosclerosis and in normal subjects.** Ann. di ottal. e clin. ocul. **79**:65-72, Jan., 1953.

40 diabetics, 96 patients with hypertension, 20 with arteriosclerosis and 88 normal persons were examined. In diabetes 62.5 percent showed aneurysms of the conjunctival capillaries, in hypertension 57.2 percent, in arteriosclerosis 55 percent and in normal subjects 36.3 percent. In diabetes the majority of the aneurysms were round, in hypertension fusiform, in arteriosclerosis saccular, and in normal persons fusiform.

John J. Stern.

## Ophthalmic Miniature

Not a more brilliant example of the value of the empirical treatment of disease is on record than the history of the application of the iridectomy to the treatment of glaucoma. Originating in what was doubtless the false interpretation of a keen clinical observation, applied to a disease which even to-day is an unsolved problem, acting in a way which von Graefe himself could not explain, and which yet lacks satisfactory explanation, it nevertheless, after 20 years of service, is the main resource in glaucoma.

A. F. Heyl,
"A contribution to the operative treatment of glaucoma,"
*Transactions* American Ophthalmological Society, 1883, p. 528.

# NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month. To receive adequate publicity, however, notices of postgraduate courses and meetings should be received at least three months before the date of occurrence.

## DEATHS

Dr. LeGrand Haven Hardy, New York, New York, died April 14, 1954, aged 59 years.

Dr. Samuel George Higgins, Milwaukee, Wisconsin, died February 9, 1954, aged 73 years.

## ANNOUNCEMENTS

### MILITARY MEETINGS

The Society of Military Ophthalmologists and the Society of Military Otolaryngologists will hold a joint dinner meeting at the time of the XVII International Congress of Ophthalmology and the annual meeting of the American Academy of Ophthalmology and Otolaryngology in New York City in September.

Dr. Frederick C. Cordes, professor of ophthalmology, University of California School of Medicine, will be awarded the plaque of honorary membership to the Society of Military Ophthalmologists at this meeting.

Cocktails and dinner will be served at 6:00 p.m. on Tuesday, September 21, 1954, in the South and Center Lounges, Shelton Hotel, Lexington Avenue and 49th Street, New York City (across the street from the Waldorf-Astoria).

All members, ophthalmologists and otolaryngologists on active duty with the Armed Forces, are invited to attend. Application should be made to Col. J. H. King, Jr., secretary-treasurer, Society of Military Ophthalmologists, Eye Clinic, Walter Reed Army Hospital, Washington 12, D.C.

### CAROLINAS MEETING

A joint meeting of the North Carolina Eye, Ear, Nose, and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology will be held at Durham, North Carolina, November 4, 5, and 6, 1954. Headquarters will be the Washington Duke Hotel.

Guest otolaryngologists will be Dr. Stanton A. Friedberg of Chicago, Illinois; Dr. John Maxwell of Ann Arbor, Michigan; and Dr. George Bayling of Durham, North Carolina.

The following guest ophthalmologists will be on the program: Dr. Charles Iliff of Baltimore, Maryland; Dr. John McLean of New York; and Dr. Townley Paton of New York.

On Wednesday preceding the beginning of the meeting, operative clinics will be held in the various hospitals of Durham and Chapel Hill.

Finally, there will be a football game on Saturday afternoon, between the University of North Carolina and the University of South Carolina, in Chapel Hill.

## SOCIETIES

### EGYPTIAN OFFICERS

At the general assembly of the Ophthalmological Society of Egypt, the following officers were elected for 1954: President, Dr. Mohammad Osman Yousef; vice-president, Dr. Ismail Disouki; treasurer and archivist, Dr. Ahmad Abd El Rehim Fahmy; secretary, Dr. Sabri Kamel; assistant secretary, Dr. Mohammad Tawfik Ismail; members of the council: Dr. Ibrahim Ahmad Mohammad, Dr. Mohyi El Din Abdalla El Arabi, and Dr. Mohammad Azmy Abd El Kadir Khalifa.

### KANSAS CITY OFFICERS

Officers of the Kansas City Society of Ophthalmology and Otolaryngology elected for the 1954-1955 season are: President, Dr. William B. Barry; president-elect, Dr. Donald O. Howard; vice-president, Dr. Barnard C. Trowbridge; secretary, Dr. Dick Underwood; treasurer, Dr. Fred Bosilevac.

## PERSONALS

The dean, Georgetown University School of Medicine, announced the appointment on April 1, 1954, of Col. John H. King, Jr., chief of the Ophthalmology Service, Walter Reed Army Hospital, Washington, D.C., to the post of clinical associate professor of ophthalmology. Colonel King will spend the summer in Europe as a consultant in ophthalmology for the Surgeon General, Department of the Army, and will visit foreign clinics for the purpose of research co-ordination.

Dr. Arthur J. Bedell of Albany, New York, has recently been elected as an honorary member of the Societe Hellenique D'Ophtalmologie, at Athens, Greece.

# The Purpose of the Guild

● The aim of the Guild of Prescription Opticians of America is to advance the science of ophthalmic optics through the development of a country-wide ethical optical dispensing service that comprehensively meets the needs of the Eye Physicians and their patients; and to educate the public to the fact that the Eye Physician-Guild Optician type of eye service truly renders the most desirable form of eye care.

*This statement of the Guild's purpose is reprinted from the cover of the REFERENCE LIST OF GUILD OPTICIANS, a geographical index of all our members. If you would like a copy, just drop a two-penny post card to*

**The Guild of Prescription Opticians of America, Inc.**

**110 E. 23rd Street New York (10) N.Y.**

Patented

***Prompt Delivery Now!***

## *MUELLER*

## *ELECTRONIC TONOMETER*

Unprecedented accuracy—*maintained* accuracy—in both tonometry and tonography—is yours with this Mueller Electronic Tonometer. Simple to use, easy to read, the Electronic Tonometer is *always* accurate. Each instrument is individually tested and certified to produce readings well within the limits established by the American Academy of Ophthalmology and Otolaryngology.

Increased production now permits immediate delivery of most orders from stock. The Tonometer, on order, can be equipped for connection of recording apparatus for tonography at extra cost. *Write today for complete descriptive booklet.* For 110 volts, 60 cycles, AC. Each, *$245.00*

***ORDER DIRECT FROM***

*V. Mueller & Co.*

330 S. HONORE ST. CHICAGO 12, ILLINOIS

## GREEN'S ELECTRIC TREPHINE

**IMPROVED MODEL BY DAVID KADESKY, M.D.**

**The instrument simplifies the operation of trephining for Glaucoma or Corneal Transplantation. Blades are provided with a shoulder that prevents penetration beyond 1.1 mm., and are available in 1½ mm., 4.5, 4.6, 5.5, 5.6, 6.5. 6.6, mm. diameters.**

**Trephine, case, and two 1½ mm. blades .....$95.00**

PARSONS OPTICAL LABORATORIES, INC.

518 Powell Street San Francisco 2, Calif.

# PRESCRIPTION OPTICIANS

ST. LOUIS, MO.

Erker Bros. Optical Co.

610 Olive Street

518 N. Grand Boulevard

and Clayton, Mo.

Prescription Opticians Since 1879

CHICAGO, ILL.

ALMER COE & COMPANY

Prescription Opticians

Established 1886

10 N. Michigan Ave.

1646 Orrington Ave., Evanston, Ill.

Dow Optical Co.

PRESCRIPTION SPECIALISTS

Suite 1015 30 N. Michigan Avenue

Chicago, Illinois

Phone RAndolph 6-2243-44

DEALERS IN OPHTHALMOLOGICAL EQUIPMENT

PORTLAND, ORE.

Hal. H. Moor, 315 Mayer Bldg.

Guild Optician

Oculists' prescriptions exclusively

NEW YORK CITY

E. B. Meyrowitz INCORPORATED

Optician Established 1875

320 Fifth Ave., New York

255 Livingston St., Brooklyn

Member Guild of Prescription Opticians of America

CINCINNATI, OHIO

L. M. Prince Co.

Established 1872

Prescription Opticians

Sole makers of Cofexic

Corrected Curve Lenses

E. B. Meyrowitz

SURGICAL INSTRUMENTS CO., INC.
520 FIFTH AVENUE, NEW YORK 36, N.Y.

LONDON *ESTABLISHED 1875* PARIS

## IMPROVED TRONCOSO GONIOSCOPE

**Dr. Manuel Uribe Troncoso, New York City**

The instrument illustrated consists of a low power binocular microscope with a magnification of 7X (though 10½X magnification is available if desired) and an illuminating telescope so mounted as to be adjustable to any focal length or type of contact glass. The illuminant is a 5.8 V Lamp controlled by a separate rheostat and for type of beam by movement of the lamp housing in a sliding mount. The light can be adjusted to a sharp image or an intense horizontal or vertical oval beam. Should the examiner wish other than fixed illumination, the lamp housing and mount may be completely detached from the scope by a ¼ turn of the locking lever.

A trigger switch in the pistol grip activates the illuminant while in use, but extinguishes it when the instrument is laid aside.

Supplied complete as described, with one large plastic Troncoso tubular contact lens and carrying case .....$325.00

*Our business was founded on these five words . . .*

"We Can Make It Better!"

*The grinding "tub"**

A little over forty years ago, N. P. Benson opened a small workshop in Minneapolis to ply his trade—grinding eye glass lenses. But there was nothing small about the ambition of this young Swedish immigrant. Even with his limited equipment, he had determined to *make lenses better than anyone else was making them!*

Since 1913 that small business has spread to 27 cities throughout the upper midwest, now employing several hundred people. Thanks to modern precision tools and master craftsmen who also believe they can "*make it better*," BENSON OPTICAL COMPANY is now a leader in quality opticianry.

**This time-honored lens grinding method has been replaced by modern surface generators in* BENSON *laboratories.*

Since 1913 Benson OPTICAL COMPANY

*Executive Offices • Minneapolis, Minn.*

LABORATORIES IN LEADING UPPER MIDWEST CITIES

## BERENS SQUARE PRISMS

These prisms have many advantages over glass
WILL NOT BREAK OR CHIP • LIGHT IN WEIGHT • LOWER COST
CAN BE REPOLISHED

These advantages make them ideal for rental sets to patients for prism exercise, are available in any combination with or without leather covered cases, prices on request.
Made in six standard sets:

No. S-4 1 each 5-10-15-20 No. S-6 2 each 10-15-20
No. S-8 2 each 5-10-15-20 No. SS-8 1 each 3-5-8-10-12-15-20-25
No. S-16 1 each ½-1-2-4-6-8-10-12-15-20-25-30-35-40-45-50
No. S-22 1 each ½-1-2-3-4-5-6-7-8-9-10-12-14-16-18-20-25-30-35-40-45-50
50

*Available at all optical and surgical suppliers.*

Manufactured by
4920 N. Lawrence St. R. O. GULDEN Philadelphia 20, Pa.
*Our new complete brochure is now available*

## THE "AMIC" MACULA DEFICIENCY TESTER

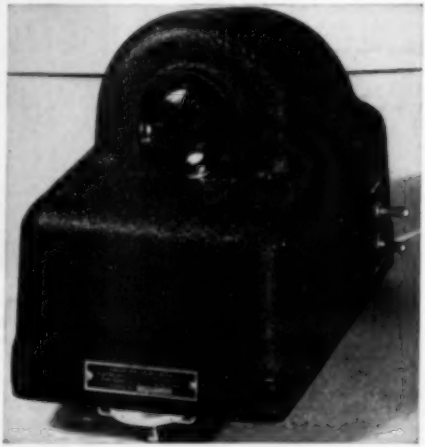
**developed by Dr. M. Goldschmidt,**
**formerly Professor of Ophthalmology, Leipzig University**

**An instrument to detect early abnormalities of the macula lutea—in a short routine check-up.**

**As a new instrument of diagnosis, it should belong to the standard equipment of every ophthalmologist.**

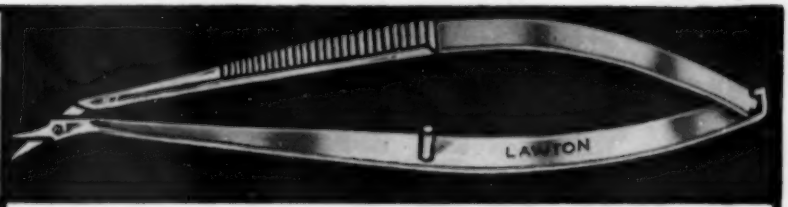
***Write for further information***

**Recently made available for immediate delivery**



**AMERICAN MEASURING INSTRUMENTS CORPORATION**
**21-25 44th Ave., Long Island City 1, N.Y.**

*McClure*
Iris Scissors
extremely delicate, short blades, angled on flat.
*Stainless Steel*
*$20.00*



# *Lawton Eye Instruments*

are made exclusively in Germany under conditions most favorable to precision workmanship. Each instrument bearing the Lawton trademark reflects the skill and knowledge of generations of expert instrument makers.

The Lawton Company offers a complete assortment of correct, new patterns, as requested by leading American surgeons. These instruments are sold through authorized surgical supply houses carrying representative stocks, and are thus made available throughout the country for your selection and immediate service.

Visit your local dealer for further information about Lawton instruments. He is well equipped to give you prompt service.

*Vannas*
Iridectomy Scissors, extremely delicate blades
Illustrated in actual size
*Stainless Steel* *$21.00*

*The* LAWTON *Company*
425 FOURTH AVENUE NEW YORK 16, N. Y.

**GREINER & MUELLER**
Expert makers
of artificial human eyes
**GLASS & PLASTIC**
55 E. Washington St. . . . Chicago 2, Ill.
Phone FR 2-4449
Branches at Kansas City, Mo., Detroit, Mich.
Our experts visit Milwaukee, Madison, Minneapolis, and St. Louis regularly. Serving the Middle West since 1924.
Eye making has been a family tradition with us since 1835.

**"LACRILENS" is a safe lens**

The test of time proves that the "LACRILENS"® contact lens can be worn for long periods of time in aphakia and in all sports, including swimming.
*Write for Brochure*

*Manufacturers of all types of Contact Lenses*

*Obrig Laboratories Inc.*
49 East 51st Street, New York 22, N. Y.

Obrig®
*Branches in* PHILADELPHIA • MONTREAL • JOHANNESBURG

# LOW VISUAL ACUITY?

**Many Cases with visual acuity as low as 2%**

**can be aided by**

*Spectel*
TELESCOPIC SPECTACLES

These successful aids to subnormal vision are available in two powers, 1.7 or 2.2 diameters. Spectel telescopic spectacles are fitted without complicated equipment or procedures. Trial sets are moderate in price.

Complete details in Bulletin 302, available from your optical supply house or direct from us.

KOLLMORGEN Optical CORPORATION

PLANT: NORTHAMPTON, MASSACHUSETTS

NEW YORK OFFICE: 30 CHURCH ST., NEW YORK 7, N. Y.

Distributed in Canada by Imperial Optical Company

*It Will Pay You*

•

to look through the advertising pages of this Journal.

We discriminate as to the quality and reliability of the advertising accepted for the

•

AMERICAN JOURNAL *of* OPHTHALMOLOGY

## "OPHTHALMIC LITERATURE"

***AN ABSTRACT JOURNAL IN ENGLISH OF THE WORLD'S CURRENT LITERATURE ON OPHTHALMIC SUBJECTS***

Indispensable to the busy practitioner for quick reference and timely useful information.

"Ophthalmic Literature" and Index (8 numbers) Yearly subscription—Four Guineas ($13.50 U.S.A.). Published by British Medical Association.

U. S. A. Agent

**GRUNE & STRATTON, INC.**
**381 Fourth Ave.**
**New York 16, New York**

# THE BRITISH JOURNAL OF OPHTHALMOLOGY

*Published monthly by*

The British Medical Association

Annual Subscription $13.50

—— • ——

# OPHTHALMIC LITERATURE

A comprehensive quarterly abstract of ophthalmology and cognate literature.

Annual Subscription $13.50

—— • ——

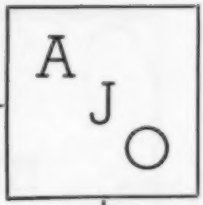
Subscriptions to:

GRUNE AND STRATTON, INC.

381 Fourth Avenue

New York 16

New York, U.S.A.




*Announcing*

TWO VOLUMES A YEAR
FOR THE SAME PRICE

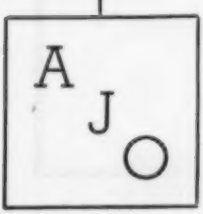
**$12.00—Domestic**

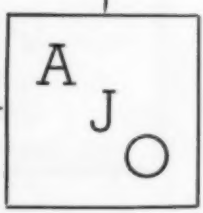
**14.00—Foreign**

Beginning with the July 1954 issue the American Journal of Ophthalmology passes another milestone by publishing two volumes a year instead of one.

Please note: The index for volume 37 was published in the June 1954 issue.

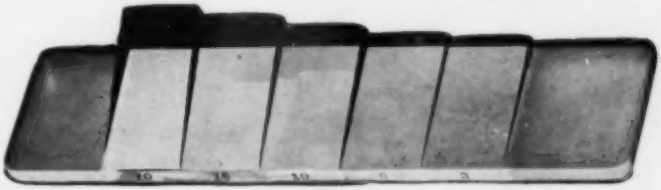
**American Journal of Ophthalmology**
**644 North Michigan Avenue**
**Chicago 11, Illinois**



# OPHTHALMIC ADJUNCTS

## *Plastic Prism Bar*



This pocket size plastic Prism Bar originated by AUSTIN BELGARD contains 3-5-10-15-20 prism diopters. Ideal for office or patients' home use.

**Each $11.00**

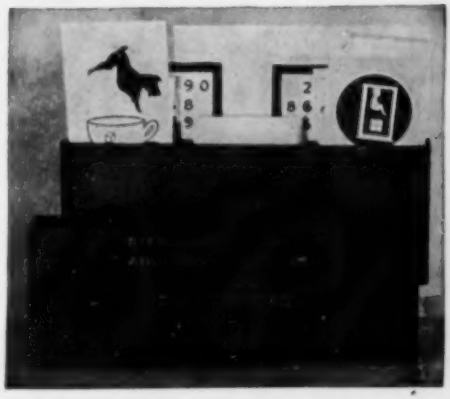
## *Stereoscopic Charts*

BY CUIDOR

Can be used with Stereocampimeter, Synoptoscope, Rotoscope and similar instruments; also with Stereoscope.

For Adults and Children.

**Set $4.75**



## *Dual Purpose Occluder & Red Glass*

This adjunct combines black plastic for occlusion and red plastic for the 9 field muscle test. Designed by AUSTIN BELGARD

**Each $5.00**

*OPHTHALMIC* Austin Belgard INC. *OPTICIANS*

WHOLESALE & SERVICE

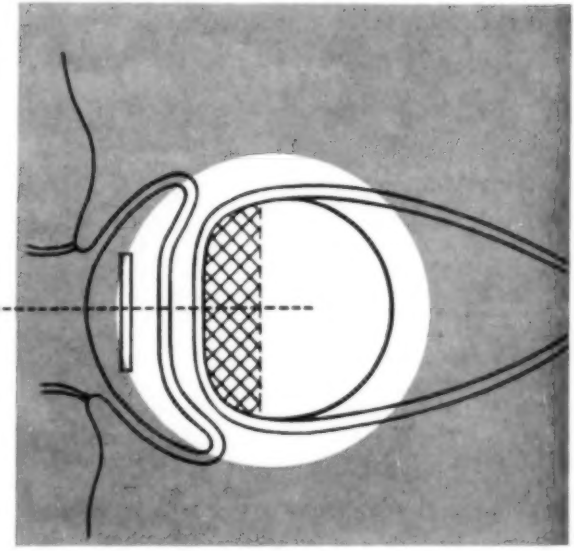
**Medical Center Office:**
**1920 W. Harrison St., at Ogden.** *(Formerly Belgard, Inc.)*

**109 N. Wabash, at Washington**
**9th Floor** **STate 2-5362**

**CHICAGO, ILLINOIS**

# Completely Contained

**within orbit**



The AO Buried Mesh Implant is designed to be completely buried within the socket. Implant will not migrate. In conjunction with the prosthesis, it serves to restore the shape and volume of the enucleated eye.

AO Buried Implants with rounded or flattened tops, have a fine tantalum mesh to which the eye muscles are attached. Either type transmits far more motility to an artificial eye than a sphere.

Available standard sizes are 19 mm., 17½ mm., 16½ mm. Other sizes on special order. Operative Procedures with AO Monoplex Implants are obtainable on request.

*AO Monoplex Laboratory will assist in the design and manufacture of special implants. Details upon request to AO Monoplex Manager, Southbridge, Mass.*

*AO Round Top Implant*

*AO Flat Top Implant*

American Optical